THE

LIBRARY

OF THE

ASSOCIATION

OF THE

ALUMNI

OF THE

COLLEGE

OF

PHYSICIANS AND SURGEONS

IN THE

CITY OF NEW YORK

SCHOOL OF MEDICINE OF COLUMBIA UNIVERSITY

ARCHIVES OF NEUROLOGY AND PSYCHOPATHOLOGY

VOLUME II

1899

STATE HOSPITALS PRESS

UTICA, NEW YORK, 1900

VOLUME II WAS ISSUED IN FOUR NUMBERS—
NUMBERS 1 AND 2 APPEARED UNDER ONE
COVER IN NOVEMBER, 1899, AND NUMBERS
3 AND 4 UNDER ONE COVER IN APRIL, 1900.

CONTENTS.

Volume II.

EDITORIAL NOTE.

Pending two years devoted to the development of the organization and sphere of the scientific work of the State Hospitals and their centre of scientific research—the Pathological Institute of the New York State Hospitals—the STATE HOSPITALS BULLETIN has served as medium of publication.

At present the plan and method of scientific investigation in the New York State Hospitals and Pathological Institute have become more defined, the lines of research of the several departments have become more completely organized, approaching more closely the original purpose of the foundation of a scientific centre of the New York State Hospitals—the plan of *correlation of sciences*, for the study of psychiatry.

This plan of scientific correlation in psychiatric research having during this period reached such a stage in its development as to unfold some definite results, it seems advisable to express the real character of our investigations, the outcome of this period of growth, in the title, more befitting the contents of the journal—ARCHIVES OF NEUROLOGY AND PSYCHOPATHOLOGY.

The ARCHIVES will contain studies on abnormal mental life and their neural concomitants, based on Psychology, Psychopathology, Experimental Physiology and Pathology, Cellular Biology, Pathological Anatomy, Comparative Neurology, Physiological Chemistry, Anthropology, and Bacteriology.

EDITORS.

DECEMBER, 1898.

VOL. II 1899 NOS. 1-2

ARCHIVES

OF

NEUROLOGY AND

PSYCHOPATHOLOGY

612.01

ON THE NUCLEOPROTEID OF THE BRAIN. (CEREBRONUCLEOPROTEID).

BY P. A. LEVENE.

[*From the Pathological Institute of the New York State Hospitals and the Department of Physiological Chemistry, Columbia University*].

Different as the activity of a nerve cell may be from that of any other cell, there are still many features common to all, and the main point of similarity is that the source of its specific peculiar energy is the substance of the cell itself, that its work is being performed at the expense of its own body. From this follows the second point of similarity, that the nerve cell cannot work forever, or any indefinite time without repairing its own substance, its own body. How does it accomplish this task? Is there in the cell a peculiar organ for that purpose or a peculiar chemical agent that is in charge of that function? Cytologists have long ago observed that when a cell is divided into two parts, so that the nucleus is left in one of them, this last part is able to recuperate from the loss and continue its life, while the other part has a

life of very short duration, and during the brief time it remains alive it does not digest nor does it assimilate food, while the first part continues to do so as well as any normal cell. Thus the cytologists have come to the conclusion that the nucleus controls the chief functions of the cell, viz.: those of repair, growth, reproduction.

Further, it is a long established fact that the predominating difference between the nucleus and the plasma of a cell is the amount of chromatin substance in them. It can be justly said the nucleus is the seat of the chromatin. Thus again biologists have come to the conclusion that the chromatin is the most important substance for the life of the cell and that most functions are connected with some changes in that substance.

If this be true, we should naturally expect to find this substance to be, first, of a very complex nature, and second, of such a nature that it can undergo different and manifold changes.

So it actually is. The chromatins belong to the class of compounds known as nucleoproteids, the most complex compounds in living matter, and probably in nature. The study of these substances in different conditions of the cell, in state of rest and activity, or better, in the state where repair predominates or dissimilation prevails, is the means of finding a clue to the solution of the problem of how the organism repairs its waste, and how we can successfully aid the organism in the most important of its tasks, when this power of restitution is for some reason or other diminished. We must remark, however, that our knowledge of the composition of these substances is not quite as extensive as is desirable, that the study of them does not date back much further than twenty years, and that least attention has been paid to the

study of the nucleoproteids (or the proteids generally) of the brain.

It was my aim to fill this gap in the study of the brain. But before reporting my results I shall recall in a few words some of the characteristics of the nucleocompounds and the main points of difference between the individual compounds of this group.

The chief characteristics are that they contain phosphorus, possess the properties of acids, and are mostly met with in combination with proteids.

The points of distinction are, first, the presence or absence of the xanthin bases in the molecule of these compounds; the character of the bases, if present; the amount of phosphorus and of proteid in the molecule, and finally the character of the proteid.

Those compounds that contain a relatively higher percentage of P and whose acidity is but little neutralized by proteids, possess a comparatively higher affinity for certain basic anilin dyes. On account of this peculiarity it was named by the microscopists—chromatin.

In order to understand the chemical changes accompanying and probably responsible for the workings of the brain it is of great interest to study the chemical changes of its chromatin in different normal and pathological conditions of the organism.

In the nerve cell chromatin is located, in distinction from many other cells, not only in the nucleus, but also in the cytoplasm (Nissl's granules), and thus naturally the question arises whether the chromatin of the latter is the same substance as is met with in the nucleus, or is it different in its nature; in other words, is there only one nucleoproteid in the nerve tissue, or more than one?

Method of Obtaining the Nucleoproteid.—As far as I know, the study of the nucleocompounds of the brain is limited to two researches, both of them quite old, dating back to the time when our knowledge of the nature of these substances and their classification was very unsatisfactory. Thus, Haliburton extracted the brain tissue with H_2O and precipitated from the extract with acetic acid a proteid containing 0.3 per cent of phosphorus. Von Jacsch treated a few human brains with pepsin–hydrochloric acid and from the residue extracted a nuclein—the nature of which he did not describe with much detail. There are a few more works dealing in a very unsatisfactory way with the general nature of the proteids of the brain, but none of them described the nucleocompounds.

The method that in my experience gave the most satisfactory results was the following:

The brains from freshly killed calves were immediately placed in alcohol-free ether and thus brought to the laboratory. After stripping the membranes, the brains were finely divided in a chopping machine and treated with large quantities of 4 per cent AmCl solution and on addition of chloroform left in well-stoppered bottles for twenty-four hours. The supernatant fluid was then decanted, and the extraction repeated with distilled water, two, three and even four times, until the extracts ceased yielding an appreciable ppt. on addition of acetic acid.

The decanted fluid was then strained through gauze and filtered repeatedly till the filtrate was perfectly clear. I have found later that the filtration is greatly accelerated, and the loss of material minimized if the strained liquid is left for several hours in separating funnels with ether. The small particles of brain tissue were then collected on the surface, and the liquid below was perfectly

clear. The filtration was thereby rendered easy. The greater part of the material, however, I worked with was obtained by simple filtration without previous treatment with ether.

The perfectly clear filtrates were then treated with acetic acid, 0.5 cc. of the acid to each 100 cc. of the liquid, and thus a precipitate of the crude nucleoproteid was obtained.

This freshly precipitated proteid is insoluble in dilute acetic acid, also insoluble in dilute hydrochloric acid, but is soluble in glacial acetic acid, in weak alkalies, as one per cent sodium carbonate, and 0.5 per cent ammonium hydrate.

It is enough, however, to let the ppt. stand over night in acidulated H_2O to lower its solubility to a very great extent, so that only a very small part of the ppt. will dissolve in weak alkalies.

The usual method of purifying nucleoproteids is to redissolve them in dilute alkalies and to reprecipitate by acids. Our proteid, for which I would suggest the name *cerebronuclcoproteid*, could not well be purified by this method, owing to the rapid loss of solubility.

There are also some objections to repeated treatment with alkalies in the fact that these might alter to a certain extent the original constitution of the proteid. For these reasons I attempted to purify the substance by repeatedly washing the ppt. first with acidulated H_2O, then with distilled water, until the latter failed to give the Biuret reaction and was free from chlorine.

There still remained the possibility that the proteid thus purified might contain some other proteids, likewise rendered insoluble by prolonged treatment with acidulated water. To ascertain whether this was the case, and also

to ascertain whether the usual method of purification affects the proteids, I endeavored to redissolve and reprecipitate some of the substance.

In doing this I encountered great difficulty in filtering the fluid, as the insoluble matter immediately clogs the filter paper. Even a constant change of the filter does not help much. In order to overcome this difficulty I recurred again to ether. The substance was treated with ether in a separatory funnel and left in it a few hours. It was then separated and filtered. The substances after they were thus purified were treated with cold alcohol, then boiled with 95 per cent alcohol, then absolute alcohol and finally with ether, until extraction was nearly complete. We found it next to impossible even after continuous extraction during several weeks to get the product in such a condition that the evaporated alcohol or ether would leave absolutely no residue.

Of the second product there was only sufficient for an estimation of the phosphorus, but a complete analysis was made of the first product.

Prep. I.

(1) 0.1675 gr. of the substance gave on combustion 0.2845 gr. of CO_2; C=42.44 per cent and 0.0987 gr. of H_2O; H=5.99 per cent.

(2) 0.2133 gr. of the substance gave 0.3615 of CO_2; C=42.28 per cent; and 0.1126 gr. of H_2O; H=5.82 per cent.

(3) 0.1415 gr. digested after Kjeldahl=0.0219 gr. of N=15.46 per cent.

(4) 0.458 gr. fused with NaOH and KNO_3 (S-free) =0.043 of $BaSO_4$, S^1=1.28 per cent.

(5) 0.3166 gr. fused with NaOH and KNO_3=0.0065 of $Mg_2P_2O_7$; P=0.573 per cent.

(6) 0.4665 gr. fused with NaOH and KNO_3=0.0092 gr. $Mg_2P_2O_7$; P=0.557 per cent.

Prep. II.

0.4897 gr. of the substance fuse with NaOH+KNO_3= 0.0078 of $Mg_2P_2O_4$; P=0.45 per cent.

	C	H	N	S	P	O
1	42.44	5.99	.....	....	....	.....
2	42.28	5.82	.....	....	....	.....
3	.....	....	15.46	....	....	.....
4	.....	....	.....	1.28	....	.....
5	.....	....	.....	....	0.57	.....
6	.....	....	.....	....	0.56	.....
	42.36	5.90	15.46	1.28	0.56	34.44

Ash=0.5 per cent.

Apparently, the first method of purification affects the proteid less than the second method, but in either case the nucleoproteid contains very little phosphorus, probably less than any other true nucleoproteid; in fact it resembles in this respect the pseudo- or para-nucleoproteids, or as Hammarsten calls them, nucleoalbumins. It was of course important to ascertain to which of the two main groups of the nucleocompounds our substance belongs, since the physiological rôle of the two is quite different.

For this purpose about 60 gr. of the substance was heated in a flask with a return condenser with 2 per cent of H_2SO_4 for about ten hours. It was then filtered, the greater part of the acid neutralized by means $Ba(OH)_2$,

filtered, the filtrate concentrated and treated in the usual way for nuclein-bases; (xanthin bases).

In the xanthin fraction but a very slight ppt. of the xanthin silver salt was obtained. So little that the attempt to obtain a xanthin reaction after the silver was eliminated, was without success.

The hypoxanthin fraction consisted mostly of guanine and adenin, no hypoxanthin being found.

Thus, it was established that our substance is a true nucleoproteid, and that two bases take part in the formation of its molecule.

Cerebronuclein.—The next task was to ascertain the cause of the low percentage of P in the nucleoproteid. This might be due to two different causes; either the nuclein itself might contain little P, or other substances might be bound to a nuclein with a high content of P, thus giving rise to an unusually complex substance.

A considerable amount of the proteid purified by the first method, but not extracted with alcohol and ether, was digested with pepsin-hydrochloric acid, for a week. The digestive fluid was then changed every two days, 0.2 per cent HCl being employed, and care being taken to have free HCl always present in the fluid. After that, the soluble products of digestion were separated by repeated treatment with acidulated water and decantation until the wash water gave no Biuret reaction, and contained no chlorine. The insoluble residue was then extracted with alcohol and ether until the latter ceased extracting, which took place after several weeks' continuous treatment. About 2.5 gr. of the pure air-dry substance was thus obtained. A small portion of it was then extracted with HCl water in order to ascertain whether it contained inorganic P; the result was negative.

The percentage of P was then estimated and 0.275 gr. of the substance fused with NaOH and KNO_3 gave 0.0140 gr. of $Mg_2P_2O_7$; P=1.42 per cent.

In comparison with other nucleins the phosphorus is seen to be rather low.

Cerebronucleic Acid.—It is known that nucleins are compounds of nucleic acid and proteids. The nuclein of the brain is exceptionally poor in P, and we are confronted again by the two possibilities that were met with in connection with the nucleoproteid itself. The low percentage of P in the nuclein might be due to the peculiar nucleic acid or to the different amounts of proteid combined with an acid having a comparatively high content of P.

The investigation in this direction is not completed at present, as we found great difficulty in obtaining a sufficient quantity of the substance.

The method of obtaining the nucleic acid that gave the most satisfactory result is the following:

The purified nucleoproteid, not extracted with alcohol and ether was dissolved in 2 per cent NaOH, while being slightly warmed on a water bath; while still warm the fluid was neutralized with acetic acid, cooled and filtered. This was found necessary for the reason that that part of the proteid which was precipitated on neutralization (alkali albuminate) was again soluble in an excess of acetic acid. The filtrate was rendered strongly acid by means of acetic acid, and was then left for twenty-four hours and filtered; to the filtrate alcohol containing 0.3 per cent HCl, was added until the fluid became very opalescent. After standing twenty-four to forty-eight hours, the precipitate was washed with acidulated alcohol, then with pure alcohol and ether, dried and weighed. The acetic solution of this

proteid precipitated albumoses and proteids from their solutions.

Seventy gr. of the proteid treated with 300 cc. of 2 per cent NaOH gave less than 100 mgr. of the nucleic acid. 0.0875 gr. of this substance fused with NaOH and KNO_3 gave 0.0105 gr. of $Mg_2P_2O_7$; P=3.35 per cent.

This P estimation can be accepted for the present only as more or less approximate to the true percentage of P in the nucleic acid.

From all these results it may be inferred that the nucleocompound of the brain is a true nucleoproteid, that it differs from other nucleoproteids by its low percentage of P, by the nature of its xanthin bases, and by the considerably high amount of proteids bound to its nuclein.

The next aim was to investigate whether the residue of brain tissue after extraction of this nucleoproteid contained another nucleocompound different in nature from the cerebronucleoproteid.

For that purpose the residue just mentioned was extracted during different lengths of time with dilute alkalies of different strengths. It was found that 0.5 per cent of ammonium hydrate will extract in twenty-four hours a considerable quantity of a proteid which can be precipitated by acetic acid, and that this is a nucleoproteid. It was also found that twenty-four hours treatment of the proteid with 0.5 per cent ammonia solution will not split off any noticeable quantity of nucleic acid. Hence, 0.5 per cent of ammonia could be applied for the extraction of the residual nucleocompounds. It remained to ascertain whether the latter was different in nature from the cerebronucleoproteid. The estimation of P in it, however, argued against such a supposition. Thus, 0.5800 gr. of

the purified substance gave on fusion 0.0105 gr. of $Mg_2P_2O_7$ or 0.5 per cent of P.

We attempted also to obtain the nuclein of that residual substance, but as the quantity of the latter in our possession was rather small, we digested the residue of forty brains with pepsin hydrochloric acid with the same precautions as mentioned above.

After the digestion and purification was completed, the residue was extracted with cold and boiling alcohol for several weeks, then with ether until the myelin was nearly extracted. In order to ascertain whether this residue contained an appreciable amount of nucleocompound, a P estimation was made.

0.4325 gr. of the substance gave 0.0140 of $Mg_2P_2O_7$, or P=0.896 per cent.

Thus, the presence of a considerable quantity of a nuclein in the brain residue was demonstrated. However, the prolonged treatment with boiling alcohol rendered the nuclein insoluble to such an extent that but little of it could be extracted by means of dilute alkalies.

Thirty gr. of the residue was treated for five hours with 0.25 per cent of NaOH and filtered directly into dilute HCl solution, a white, floculant ppt. was formed, but in a quantity not sufficient for further analysis.

No marked difference between the residue and the cerebronucleoproteid could be found in the character of their xanthin-bases. Here, again, guanin was found to predominate, the other bases not being sufficient in quantity to be identified.

All these results do not bear out the supposition of the existence of more than one nucleoproteid in the nerve cell.

From these results it may also be inferred that the

nature of the chromatin of the cytoplasm does not differ from that of the nucleus. However, this question can be fully elucidated only by a comparative chemical study of the nerve tissue under different physiological and pathological conditions; *i. e.*, in conditions when the chromatin nearly disappears from the nucleus and is located only in the cytoplasm and vice versa.

I wish to acknowledge my indebtedness to Prof. Chittenden for his valuable suggestions and for the privileges accorded to me in the Laboratory of Physiological Chemistry of Columbia University.

MAY 20, 1899.

REFERENCES.

HALIBURTON: *Journal of Physiol.*, Vol. XV, 1893.
HAMMARSTEN: *Zeitschr. f. Physiol. Chem.*, XIX.
V. JACSCH: *Pflugers. Arch.*, Vol. XIII, p. 469.

612.01-615.2

IODINE-COMPOUNDS IN THE TISSUES AFTER ADMINISTRATION OF KI.

By P. A. LEVENE.

[*From the Pathological Institute of the New York State Hospitals*].

The mechanism of the action of most of the drugs is as yet quite obscure. All that is known is their final effect on the organism, and not the process in the cells and tissues leading to that effect. Not much fuller is our information of the course of the affinity of certain drugs to certain organs and tissues. Why are not all the tissues equally influenced by the same drug, and why are not all animals equally affected by the same drug,—why is one animal refractory to a substance which is a strong poison to another? The works of Ehrlich and Metchnikoff and their followers on one hand and of Overton on the other, have greatly enlarged our knowledge of the subject, but with their work our knowledge only begins, and there still remains more dark points than clear ones.

Already *à priori* it would not seem probable that a drug could produce in a cell, tissue or organ a function they were not performing normally. What could be expected of a drug is that it would act as an agent stimulating or depressing all or certain functions of a cell or organ,—which it may do in two different ways, either changing what may be termed the physical conditions of the tissue, or forming new compounds with the cell and tissue constituents.

Our aim was to investigate whether such new compounds are actually formed, and if so, in what tissues.

We decided to begin the experiments with KI, as the latter is and always was a favorite remedy in the hands of the neurologist, and then within the last few years there have been found in different organisms and organs many compounds of I with such substances as were generally met with in other tissues I free.

Drechsel was the first to find I in the keratin Gorgonia Cavaloni; then Baumann found his thyroiodin, etc. Later Drechsel found that the hair of individuals that had been taking KI contained I in the form of iodofat. Iodofat was also found by Winternitz in the bones of animals fed on KI.

Finally many normal cell constituents can be combined with I artificially outside of the organism. It was natural to question whether or not all tissues and cells possessed the same ability as the thyroid gland, the hair and the bones.

My experiments were carried out on hens that received every morning during one week 1 gr. of KI, and later a higher dose of 2 gr.

My object in selecting the hen for the experiments was that here I could have at least one organ or tissue on which we could study the changes from day to day, and thus we could see not only the final results, but probably also the intermediate stages.

Further, here we could examine cells that were developing while the animals were, so to say, saturated with I salts, and, lastly, eggs offer a very convenient material for isolating the different chemical cell constituents.

Thus the investigation naturally falls into two parts, first, that of the eggs, collected while the birds were fed on KI, and, secondly, that of the tissues of the hens at the end of the experiments.

I.

METHODS.

As related above there were found in different organs and organisms compounds of I with proteids and related substances, with fat, and I in the form of mineral salt. In this work all these three constituents of the egg were examined for I.

In order to test for the iodides the proteids of the eggs (the white and the yelk were always treated separately), were precipitated by coagulation by heat, and a few drops of acetic acid, or by means of sulphate of ammonia, and the filtrate tested for KI.

To test for iodo-proteids, the eggs were extracted with cold and hot alcohol until the last ceased extracting any I containing substances, the residue was then fused either with NaOH and KNO_3 or with a mixture of one part of Na_2CO_3 and two parts of KNO_3; the fusion dissolved in H_2O, filtered, sulphurous acid added and tested for I.

Test for iodofat.—The tissue was extracted with alcohol. The extract evaporated, the residue extracted with ether and left for twenty-four hours, then filtered and washed in a separatory funnel with H_2O until the last showed no trace of I.

The ethereal solution was then treated in two ways: first, the ether evaporated in vacuo, the residue saponified by means of alcoholic soda, the soap then tested for I by fusion. The soap was further decomposed by means of weak acid, and I searched for in the filtrate and in the residue.

Second, the ethereal solution was treated with freshly prepared sodium alcoholate and left for twenty-four hours. It was then filtered on a suction pump and washed with

ether until the washings seemed colorless. The washings were evaporated and tested for I by means of fusion.

The soap was decomposed by means of acid, and I searched for in the fatty acids and the filtrate.

II.

RESULTS OF THE ANALYSIS OF THE EGGS.

About four eggs were taken for each experiment, and the experiment repeated several times. Only iodides could be detected.

The results remained the same for about three weeks. Once after the proteids were separated by means of heat coagulation on addition of acetic acid the filtrate showed the chloroform test on addition of dilute nitric acid.

After the third week the hens ceased laying eggs for about two weeks and began at the end of the fifth week. The eggs collected during the sixth and seventh weeks were again subjected to the tests described above.

Iodides were found present in the white and yelk, no iodoproteids in either of them.

The analysis of the ethereal extract washed with water until the water ceased extracting I gave the following results:

One part was saponified with alcoholic soda, the soap washed with ether, the ethereal extract evaporated, fused and tested for I with negative results. The soap on fusion gave a positive test for I; when decomposed by dilute acid no I was found in the fatty acids, but I was present in the filtrate of the fatty acids. In one case the filtrate was divided into two equal parts, one tested for I directly, the other evaporated to dryness, fused and then tested for I it seemed to give a more intense color with

the same quantity of chloroform. No quantitative determination, however, was made.

The second part was saponified by means of freshly prepared sodium alcoholate, left over night, then filtered and washed with ether. The filtrate and ethereal extract evaporated, fused as mentioned above and tested for I with positive results, the soap was then decomposed by means of H_2SO_4, the fatty acids contained no I nor could it be detected in the filtrate of the acids.

Howald, who employed the same method, never found any I in the ethereal washings of the soap, nor could he detect any in the filtrate of the fatty acids.

Our experiments with fatty acids and fats into the molecule of which I was introduced, did not give such positive results as Holmes claims. This question, however, requires further study.

III.

ANALYSIS OF THE TISSUES OF THE HENS.

The following organs and tissues were examined:

The nervous tissue.

The muscular tissue.

All the glandular organs taken together.

Gastrointestinal tract.

Skin and adipose tissue.

Bones.

The methods of analysis were the same.

Should it be true that halogen fats are so readily saponified by sodium alcoholate and on the other hand, that the soaps of those fats on decomposition with dilute acids do not lose their halogen, then the organic substances found in the yelk of the egg must be some other organic compound, not a fat.

As mentioned above, iodides could be found in nearly all the organs and in greatest amount in the intestinal tract (probably the part not yet absorbed) and in the bones. No iodoproteid could be detected in any of the organs.

The ethereal extract of the bones treated as above, showed the presence of I, which agrees with the findings of Winternitz.

It should be mentioned also that Holmes found I combined with the keratin part of the hair, and it seemed to us possible to find it also in the keratin of the nervous system, but analysis did not corroborate this anticipation.

Thus it appears that seemingly the same tissue constituents respond differently to ingested KI. Whether this depends on a difference in their chemical constitution at present unknown to us, or whether the cause of it lies in the peculiar function of certain organisms, organs and tissues, this will have to be answered in the future.

May 20, 1899.

BIBLIOGRAPHY.

Baumann: *Zeitsch. f. Physiol. Chem.*, Bd. XXI and XXII.

Drechsel: *Zeitsch f. Biologie*, 33, 85, 90.
Centrbl. f. Physiologie, 9, No. 24.

Howald: *Zeitsch. f. Physiol. Chem.*, XXIII.

Winternitz: *Zeitsch. f. Physiol. Chem.*, XXIV.

THE CRANIAL AND FIRST SPINAL NERVES OF MENIDIA; A CONTRIBUTION UPON THE NERVE COMPONENTS OF THE BONY FISHES.

By C. JUDSON HERRICK,
Associate in Comparative Neurology, Pathological Institute of the New York State Hospitals.

Section 1.—Introductory.

I.—General Introduction.

Looked at from the biological standpoint, the primary function of the nervous system is to put the organism into relation with the outer world, the external environment. The health of the body is largely determined, in last analysis, by the perfection of the correspondence between the inner activities and the forces of nature outside.

The immediate mechanism upon which this correspondence directly depends—the sense organs and peripheral nervous system—must not be overlooked by the neurologist who would thoroughly understand the normal or pathological processes of the nervous system as a whole. Even the higher psychic processes in the central nervous system cannot be thoroughly understood without the knowledge of the peripheral nervous system. This is clearly understood by the psychologists who recognize that psychic phenomena cannot be studied without reference to their physiological concomitants, which in turn requires investigation into the morphology of the nervous system both peripheral and central.

It is unnecessary at this late day to urge the importance to normal physiology (and not less to pathology) of a true

Note.—This study was awarded the Cartwright Prize for 1899 by the Alumni Association of the College of Physicians and Surgeons, Columbia University, New York.

morphological comprehension of the structure that reacts to stimuli of the external environment. This implies a broad philosophical appreciation of the relations between part and part; of the functional, mechanical and other factors which determine the forms of the parts; of the modifications induced by the mechanics of growth during the ontogeny; and, finally and most important of all, of the phylogenetic history. It is this latter point which most often gives the clue to structure, and this is a justification for the recognition of comparative anatomy in a scheme of the correlation of sciences for the study of the dynamics and statics of the nervous system.

A generation ago comparative anatomy in this country was chiefly in the hands of the medical profession, and the medical journals contained many memoirs upon the anatomy of the lower animals, memoirs that are standard sources of information to the biologists to-day. With the development of medical specialties and the advance of specialization in other departments of knowledge, all this is changed and it often happens that the pathologist of to-day, for instance, is acquainted with the normal structure of the organs in the human body, the morbid processes of which he is investigating, but knows little of their comparative anatomy, histology and embryology. Of course it is not to be expected that under the present conditions pathologists should conduct special researches in comparative anatomy or embryology; nevertheless an acquaintance with the general principles of these subjects is indispensable for pathology if this science is to gain a broader and more comprehensive basis.

How much would be left of the general laws of the science of embryology if all of the facts acquired by the comparative method were stricken out? And if physi-

ology had never availed itself of the opportunities afforded by experimentation upon the lower animals, as a science it would be more than emasculated; indeed it is doubtful if it would ever have been born.

Neurology, not less than these other two sciences, is dependent upon the comparative method for its guiding principles, though the medical profession as a whole has been slow to seize the opportunities thus afforded. Any one who will take the trouble to examine the instruction in the anatomy of the brain in many of our medical colleges (and until very recently in some of the best of them) will find the justification for this latter remark. A glance at the works of Edinger and the other apostles of the comparative method should convince the most skeptical that it is impossible to understand, much less to teach intelligibly, the complexity of the adult human brain without reference to the simpler and more diagrammatic types presented by the lower vertebrates. This is undoubtedly the most valuable advantage to be derived from the study of comparative neurology.

From our standpoint, however, in a system of correlated sciences for the study of abnormal mental and nervous life the most important function of comparative neurology lies in its value as a method of research. Of primary importance in the investigation of the phenomena in this domain, as in all life phenomena, is the method of pathology. By the use of this method the phenomena restricted within the limits of the normal are given a wider range, are magnified. It is obvious that we are thus enabled to get a deeper insight into the nature of the phenomena and have a broader basis to form inductions which are more secure in proportion to the extent of territory from which they are drawn.

The pathological method is one of experiment; indeed pathological conditions are all experiments—nature's experiments—ready at hand for investigation and often more ingenious than we could invent. The great power of the pathological method is that phenomena are often simplified by being split up into their components. A set of greatly complicated phenomena is detached from the others, is dropped out of the intricate series, becomes isolated and thus amenable to control. Having before us isolated and accentuated components of the intricate phenomena forming pathological processes, we can often by means of experiment modify these detached factors and still further analyze them into simpler elements. Finally by studying different phases of a pathological process the component factors of a phenomenon are reduced to simplest terms, analyzed into elementary units. This accomplished, we may undertake a synthesis of the factors, arranging them with relation to cause and effect, and thus arrive at a formula or generalization which will not only explain the single phenomenon, but also all other phenomena of the same kind. The pathological method, then, stands foremost in scientific investigation of organic phenomena, even of the normal manifestations.

Comparative neurology, like all other sciences that deal with life phenomena, must use similar methods, among which the methods of experimental pathology play an important *rôle*. But comparative neurology uses largely the pathological method, in the wider sense of the term; for in the nervous systems of the lower animals we perceive again and again the analogies of pathological conditions with the only difference that they are spread out in time and extended along the phylogenetic pathway

instead of being concentrated and occurring in cataclysmal fashion as, for instance, in nervous or mental disease in the human subject. Thus, when an animal changes the aquatic for a terrestrial habitat, with the disuse of aquatic sense organs and their substitution by those fit for terrestrial environment, we have practically an example of the pathological method in the atrophic process occurring in the conducting tracts of the sense organs that fall into disuse. Perhaps we might also compare the hypertrophy of the cervical cord in the sea-robin, or that of the vagal lobes in the carp to the hypertrophies occurring as a response to increased function in pathological processes.

The very fact that the natural experiments in the nervous systems of the lower animals are spread out in time gives the method of comparative neurology a particular value, indeed a value not possessed by other methods of investigation in mental and nervous life. For, the terms of the series of the evolutionary process modifying the reactions and structure of the lower forms of the nervous system being extended over great periods of time and taking place exceedingly gradually, the integral phases of the process are obtrusively unfolded. Whereas in disease of the human subject or pathological manifestations induced in an individual organism the process occurs so rapidly that its serial phases are run together and the progression of the terms of the process eludes one's grasp.

I would emphasize the importance of comparative neurology as a *method of research* in an organization of sciences for the investigation of the phenomena of consciousness and their physiological concomitants. This department of science from this standpoint should be turned into account toward the solution of problems of

the general laws of the dynamics and statics of the nervous system as a whole, rather than towards contributing merely complete knowledge of morphology or even specific physiological data of a particular organism and stopping then and there without probing into the general relations of one particular nervous system to all other nervous systems.

We should study the nervous system of any particular animal or species in a determinate fashion, with the purpose of interpreting structure in dynamic terms and of throwing light upon the nervous system universally and upon the inter-relation of the phylogenetic and ontogenetic progression, bringing comparative neurology into correlation with biology, physiology, pathology and the psychological sciences. Again, should we find in the neurons of some of the lower animals, peculiarities of reactions to stimuli, or variations in growth or metabolism, evident in function or structure which would throw light upon the life history of *all* neurons, and should we use this knowledge in the form of a generalization, we would then be using comparative neurology in its proper sphere in the co-ordination of sciences for the purpose of determining the nature and laws of the nervous system.

It seems to me that the rôle that comparative neurology plays in the correlation of sciences is primarily in its use as a method, or instrument, keen in investigation of the nature of the nervous system. Comparative neurology is a means of verification, a control to the deductions gained from the pathological method proper, which in my opinion is the most powerful method in the investigation of *normal* life phenomena.

This brings us to the motive of this paper. What has it to do with psychiatry? The answer to this is very simple.

It has now become a commonplace in science that all parts of the nervous mechanism are so intimately interwoven and interdependent that the study of the functions of the lower parts of the human nervous system is indispensable for a correct comprehension of the higher spheres of the brain.

Our aim in this paper is to lead up to the solution of the functions and structure of the cranial nerves and their intra-axial continuations not only in the lower and higher vertebrates but also in man. With a few notable exceptions, anatomists of the human nervous system have failed to bridge over the gap between their own field and that of the comparative neurologists, especially as to a comprehensive study of cranial nerve morphology and physiology. The same may be said of the comparative neurologists. Hence the subject is full of confusion. Our aim is to bridge over this gap and ultimately not only to contribute to the knowledge of the human nervous system in particular, but also to work out some of the general laws of structure and function of the nervous system in general.

Let us now, although in an introductory manner, consider more specifically the advantages of comparative neurology as a method of research and some of its bearings upon our problem of the phylogenesis of the cranial nerve components.

II.—Introductory Sketch of the Theory of Nerve Components.

One of the most striking of the recent neurological findings has been the discovery of the history of the cerebral cortex. Starting from the fish types, in some of which the pallium is a simple non-nervous membrane, it is interesting to watch the emergence of the cortex and parallel with it the progressive advance in psychomotor

manifestation, as one sense after another effects its secondary connections with the cerebrum. Thus the fluctuations of the relative importance of the organs of higher sense, as the nose and the eye, from type to type are clearly reflected in the size and organization of the corresponding primary and secondary brain centres. We have, it is true, as yet only a few hints in these directions; but yet enough has been gained to illustrate the exceeding fruitfulness of this line of research.

Now, in the domain of the peripheral nerves we have as yet developed but few such illuminating generalizations, and our students still memorize the twelve pairs of cranial nerves, their trunks, rami and ramuli, with the distribution of each, much as one would learn a Greek paradigm. If there is any morphological nexus between the various nerves or any basis for a rational classification, the average text-book gives no hint of it.

In view of the present inchoate condition of the morphology of the cranial nerves and of the fundamental relation of this problem to the proper understanding of the great afferent and efferent systems of the neuraxis itself, it is most fitting that within very recent years there has been a notable increase in the number of researches centering about these questions.

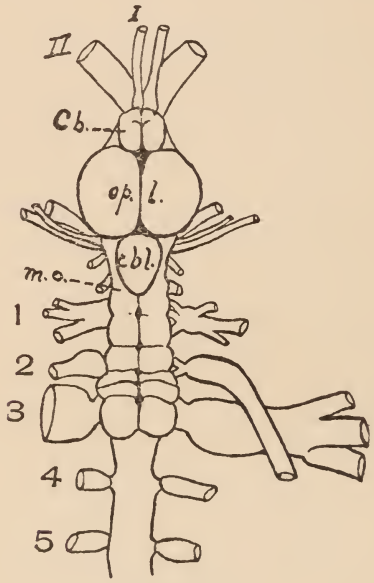
The literature of the cranial nerves is remarkably voluminous, but by far the larger part is either purely descriptive or dominated by crude and false morphological theories. It is only within the present decade that a really practical standpoint has been introduced for the proper morphological treatment of the cranial nerves—at least their sensory portions. This is the doctrine of nerve components, which had been earlier applied to the spinal roots in the very suggestive "four-root theory," and which

now affirms that in a similar way the cranial nerve trunks may contain several varieties of sensory fibres which have different functional and morphological relations and several of which may be present in a single segmental nerve. Since these systems of components are defined by both the peripheral and central relations of their fibres, it is obvious that the ordinary methods of research are inadequate for their study, since these methods have usually examined the proximal termini microscopically and the peripheral courses macroscopically, with but slender basis for an exact correlation of the two sets of findings. The only case thus far published in which both central and peripheral relations of the sensory components have been fully worked out in the same type is Strong's research ('95) upon the cranial nerves of the tadpole of the frog.

Numerous other students, both in this country and in Europe, are now at work upon different phases of the problem, and this activity is expended mainly upon the fishes. The reasons for this are evident, for not only do these primitive types present the problem in its simplest terms and in terms easily assimilable to the paradigm given by Strong, but the extreme diversity among the various groups of fishes in the relative development of the several nerve systems gives us a remarkably beautiful morphological series which sheds much light upon the relationships of the components.

Nature has, as it were, performed for us in the fishes a series of experiments which reveal as clearly what are the primary and secondary anatomical centres for the several systems of sense organs as the experimental method of v. Gudden or even this in combination with the Nissl stain. The working out of the details of this scheme proves more difficult than would at first sight be supposed

and our knowledge of the exact relations in the fishes is as yet very fragmentary. Nevertheless enough has already been done to indicate the broad lines along which these correlations are to be looked for and we may now associate the several lobes of the medulla oblongata which are so characteristic of the fishes with their respective cutaneous or visceral sense organs as definitely as the olfactory nerves are associated with the olfactory lobes or the electric lobes of the torpedo with the electric organs.



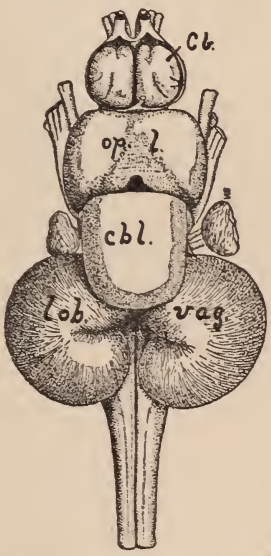
TEXT-FIGURE 1.

TEXT-FIGURE 1.—Brain and part of spinal cord of *Prionotus* seen from above. After Morrill. The spinal nerves are numbered in Arabic numerals. *Cb.*—cerebrum; *cbl.*—cerebellum; *m. o.*—medulla oblongata; *op. l.*—optic lobes.

Moreover the homologies of these lobes among the different groups of fishes and in the higher vertebrates, even up to the human brain, can now be followed with considerable precision, as we shall see beyond.

One of the best illustrations of this central response to peripheral differentiation is found in the sea-robins, in which certain free rays of the pectoral fins have become exceedingly sensitive finger-like tactile organs and their sensory nerves, together with the corresponding dorsal horns of the spinal cord, have been in consequence enormously hypertrophied.

This brain is strictly typical for the bony fishes, but the proximal end of the spinal cord exhibits a series of remarkable lobes which are the terminal centres for the



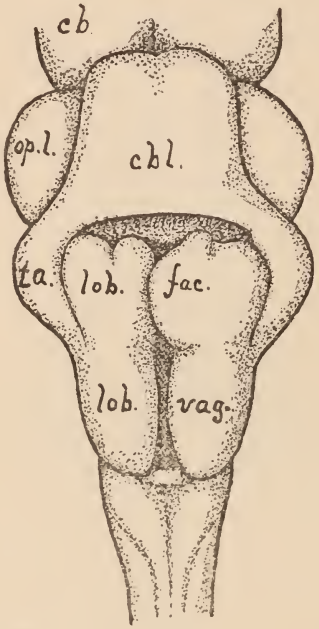
TEXT-FIGURE 2.

TEXT-FIGURE 2.—Brain of *Carpiodes* as seen from above. After C. L. Herrick. *Lob. vag.*—lobi vagi; other letters as in fig. 1.

sensory nerves from the free rays. (Text-figure 1). Here the exaggeration of the general cutaneous component of these first three spinal nerves evokes a perfectly definite and easily recognizable response in the central system.

No new component is introduced; a pre-existing structure is simply enlarged.

Again, one of the most conspicuous features of the brain of certain types of fishes, such as the carp, is the enormous lobi vagi. (Text-figure 2). These constitute the terminal nuclei of the vagus nerves and correspond, at least roughly, to the sensory vagus nuclei of man. They are very small in fishes like the eel, whose gills are



TEXT-FIGURE 3.

TEXT-FIGURE 3.—The brain of *Amiurus* as seen from above. After Kingsbury. *Lob. fac.*—the lobus facialis; *ta.*—the tuberculum acusticum; other letters as before.

reduced, but in the cyprinoid fishes are related not only to the elaborate gill apparatus and the taste buds of the mouth, but especially to the buds of the huge and peculiar palatal organ and also to the widely scattered

sense organs of like nature all over the skin of the head and trunk, the so-called terminal buds. Fishes generally have an elaborate system of taste buds in the mouth, all of which are related to this centre; but they differ widely in the number of terminal buds on the outer skin, and in all known cases the size of the lobi vagi is increased where these latter organs are numerous.

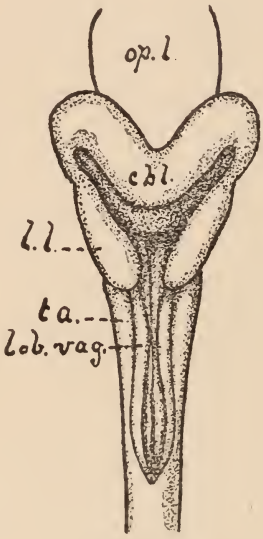
The cat-fishes and some others present an interesting modification of this case. Here the terminal buds which are supplied by the vagus nerve, are reduced in number, but on the head and especially on the barblets, these organs are exceedingly numerous. Accordingly, the branches of the facial nerve which supply these regions are enlarged and a terminal centre in front of the lobus vagi is developed for them, the lobus facialis (the so-called lobus trigemini of the older authors). This lobus is only a pre-auditory derivative of the lobus vagi and all nerves related to these two homodynamous centres can be treated as a morphological unit, the "communis system." (Text-figure 3).

These lobes constitute visceral or special sensory centres and can in no respect be compared with the dorsal horns of the spinal cord. They are new structures developed in the head in correlation with distinctively cranial sense organs. If represented in the trunk at all, it could only be in the feebly developed visceral sensory centres of the spinal cord.

The cat-fish illustrates another one of the medullary centres, whose size is exceedingly variable among the vertebrates. This is the tuberculum acusticum, in man related only to the auditory nerve, but in the fishes serving as the terminal nucleus of the entire lateral line

system of sense organs as well (Text-figure 3, *ta.*). A comparative study reveals the fact that this centre varies in size with the degree of differentiation of the lateral lines.

Thus, in the sturgeon, whose other medullary centres are relatively feebly developed, this system is greatly enlarged and a portion of it has been differentiated in front to form the lobus lineæ lateralis of Johnston (Text-figure 4, *l. l.*), the so-called lobus trigemini of the older authors.



TEXT-FIGURE 4.

TEXT-FIGURE 4.—The brain of the sturgeon, *Acipenser*, as seen from above. After Goronowitsch. *l.l.*—lobus lineæ lateralis; other letters as before.

This acustico-lateral centre, like the vagal lobe, is difficult to correlate with anything found in the spinal cord.

These cases, and many others which might be given, illustrate the way in which comparative anatomy assists

us in the analysis of the sensory components of the cranial nerves by the comparative study of the correlated variation between the sense organ and the centre. It should be remembered, too, that these variations in the primary centres involve corresponding changes in the secondary tracts and centres, and thus contribute to some of the most difficult problems of morphology. Much remains to be done in working out these principles; it is, moreover, the most sound morphology, and the most economical as well, to continue this line of research among the fishes until its most fruitful suggestions have been gathered in before attempting the application in detail to human anatomy, for we find no other group of the vertebrates so diversely specialized in these respects as the fishes, and hence presenting so varied an assortment of stages in the development of the several systems of components.

The research which follows is an attempt to solve some of these problems in the fishes, and it is believed that the results will contribute something toward the development of a true philosophical understanding of the human nervous system. The author feels that at the present time the most important step in this direction is a clear insight into the nature of the several components of the cranial nerves, their relations to each other and to the spinal nerves, and particularly their significance to metamerism and the allied head problems. Until each component can be isolated and treated as a morphological unit and then unraveled in its peripheral courses through the various nerve roots and rami—until this is possible no further great advances in cranial nerve morphology can be looked for even among the lower vertebrates, still less in man. For in the human subject whole systems of nerves have been

dropped out, new ones have been added and primitive relations have been distorted by the usurpation of vast areas of the head by nerves of distant segments. It is to this problem that the author has addressed himself primarily in the following pages.

The doctrine of nerve components dates properly from the systematic separation of sensory and motor roots and the formulation of Bell's law. Gaskell's suggestive "four-root theory" has been a stimulus to further advances and now it is customary to recognize in the spinal nerves of the vertebrata four types of fibres: (1) somatic efferent and (2) somatic afferent (general cutaneous), making up the major part of the ventral and dorsal roots respectively, and (3) visceral efferent and (4) visceral afferent. It is probable that the visceral efferent fibres go out with both roots and the visceral afferent enter by the dorsal root.

The proper analysis of the cranial nerves has been retarded by various uncritical attempts to conform them rigidly to Bell's law. These attempts resulted only in confusion so long as qualitative differences other than sensory and motor in the nerve fibres were not recognized and all sensory cranial nerves were compared directly with dorsal spinal roots (general cutaneous), and all motor cranial nerves were compared with the somatic motor fibres of the trunk. Our precise knowledge of the sensory components in the cranial nerves of the lower vertebrates begins with Strong's paper on the Cranial Nerves of Amphibia ('95), and the present research was carried out upon the basis of that work.

Throughout the Ichthyopsida we can at present distinguish in the cranial nerves three sensory systems of components and two motor, aside from the sympathetic. Each

system may be defined as the sum of all fibres in the body which possess certain physiological and morphological characters in common, so that they may react in a common mode. Morphologically, each system is defined by the terminal relations of its fibres—by the organs to which they are related peripherally and by the centres in which the fibres arise or terminate. The fibres of a single system may appear in a large number of nerves, repeated more or less uniformly in a metameric way (as in the general cutaneous system of the spinal nerves), or they may all be concentrated into a single nerve (as in the optic nerve). The post-optic systems are as follows:

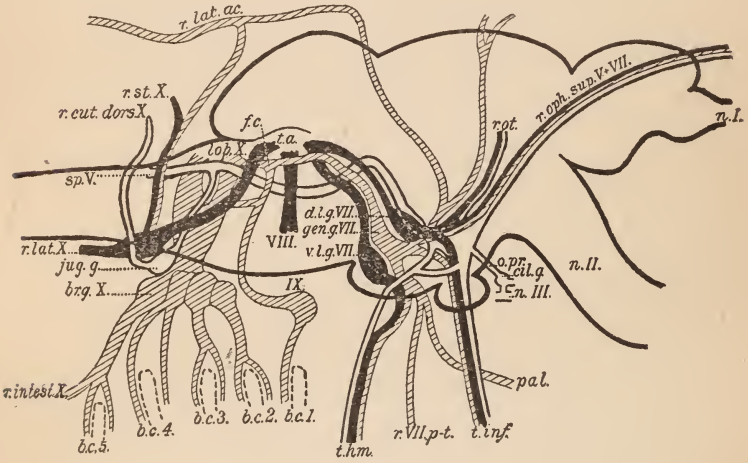
(1). The *general cutaneous system* of the head is clearly the representative of the somatic afferent of the trunk. Its fibres innervate the skin in general, without specialized sense organs and all terminate in the spinal V tract or cells associated with it, these being the continuations into the head of the dorsal horns of the spinal cord.

The term *somatic sensory system* has been used for these components. This usage, however, is ambiguous. Gaskell used this term for all nerves to the outer skin, as distinguished from visceral nerves. Cole would exclude the lateralis nerves, but apparently would include nerves for terminal buds of the skin, which we would exclude as belonging to the communis system. It is better, therefore, to avoid the use of this name altogether.

(2). The *communis system* innervates visceral and mucous surfaces, taste buds and other similar specialized end organs (terminal buds) on the outer skin which do not belong to the lateral line system. These fibres are distinguished by their fine calibre and by the fact that they all end in a single centre, the lobus vagi (which has, however, in some teleosts a specialized pre-auditory portion, the so-called lobus trigemini). They may enter this

centre directly through the vagus roots or indirectly through the fasciculus communis from the IX or VII roots.

I use the term *communis system* in preference to the "fasciculus communis system," proposed by Strong and subsequently employed by Allis, Kingsbury and myself because the system contains many fibres which do not enter the fasciculus communis in the strict sense of that term. Moreover the non-committal character of the shorter term relieves it of the ambiguity which has of late arisen regarding the morphology of the fasciculus communis—see the account in Section 3.



TEXT-FIGURE 5.

TEXT-FIGURE 5.—A diagrammatic view of the sensory components of the cranial nerves of Menidia, as seen from the right side. The diagram is based upon the projection of the cranial nerves upon the sagittal plane which is given in detail on figs. 3 and 4. The general cutaneous component is indicated by the unshaded nerves, the communis component by cross-hatching and the acustico-lateral is drawn in black. For the significance of the reference letters see the table of abbreviations at the end of this article.

(3). The *acustico-lateral system* innervates the lateral line organs and the internal ear. Its fibres are for the most part very large, the coarsest in the body, and they

terminate mainly in the tuberculum acusticum of the medulla oblongata.

(4). The *somatic motor fibres* are commonly regarded as represented by the eye-muscle nerves, the III, IV and VI pairs, and in higher forms by the hypoglossus nerve. In the fishes the latter is a true spinal nerve.

(5). The *viscero-motor fibres* comprise the motor roots of the V, VII, IX and X nerves.

No cranial nerve contains all of these components and it is not necessary to assume that any one ever did contain all of them, for some of these components are evidently neomorphs in the head. Their relations and probable morphological significance are discussed in Sections 3 and 12. This brief outline, together with the accompanying text-figure and figures 3 to 5 and 8 to 12 will perhaps aid in rendering the descriptions which follow more intelligible. Somewhat fuller general accounts have been given in my preliminary papers ('97 and '98a).

III.—Scope and Methods.

This investigation was begun as occupant of the Columbia University Room at the Marine Biological Laboratory at Woods Hole, Mass., in the summer of 1896, was continued during the winter of 1896–7 in the Zoological Laboratory of Columbia University, and subsequently at the Pathological Institute of the New York State Hospitals and at the laboratory of the U. S. Fish Commission at Woods Hole. To all of these institutions I am under obligation, not only for the facilities of research, but for numerous special courtesies; and particularly to Dr. Strong of the Columbia Laboratory for suggestion and advice throughout.

The main object of the study has been, as suggested above, the analysis of each of the cranial nerves, especially the sensory portion, into its components and the tracing of these components continuously from their nuclei in the central nervous system to their ultimate peripheral distributions. As this has necessitated the careful microscopical study of the entire courses of these nerves, it has seemed best to examine and plot the entire peripheral nervous system, even those nerves which, like the pure motor branches, do not bear directly upon this major problem. The following pages, therefore, aim to give a measurably complete account of the cranial and first spinal nerves of the type chosen for the detailed analysis. This mode of treatment receives further justification from the fact that the nervous system of no member of the family represented by this type has ever been described, even topographically, so far as my knowledge goes.

The descriptive details thus accumulated, together with bibliographical and critical comments upon them, so far as they may be of value to special students of comparative anatomy, are given with considerable fulness. A summary at the close of each section and an analytical table of contents will assist the general reader who may not be interested in such details.

The reconstructions plotted upon Figures 3, 4, 5 and 7, all illustrations of cross sections of Menidia, and the diagrammatic cross sections, Figures 8 to 12, are all based upon a single series of sections, and the descriptions in the text have, for the most part, been written from this same series. Thus individual variations do not enter into the descriptions. Other specimens prepared by the same and by different methods have, however, been used

constantly for control and important variations are noted in the text.

The plots are accurately drawn to scale, the magnification being indicated in each case, so that measurements in the text are, as a rule, unnecessary. The figures on the scales in the margins of the plots indicate the serial numbers of the sections plotted, the sections being in all cases fifteen micra thick. To facilitate the location on the plots of objects mentioned and to avoid the introduction in the text of detailed measurements, these serial section numbers are often referred to in the descriptions.

The genus Menidia contains two species, both of which are commonly called "silver-sides." They are very abundant near Woods Hole, Mass., swimming in close schools in shallow water, the larger one, *M. notata*, being found along the open shores, while the smaller one, *M. gracilis*, is more commonly taken in the protected bays and tide pools. They are both small species, the larger one being about 10 cm. long. Their food in captivity seems to be mainly small crustaceans, such as the megalopa larva of the common crab, though they eat smaller fishes of their own or other species.

Regarding the relationships of the family Atherinidæ, to which they belong, I am permitted to make the following quotation from a personal letter received from Dr. Theo. Gill. "The Atherinids, I think, are a primitive type of Acanthopterygian fishes, near the border line between ordinary physostomous and physoclistous fishes. They are genetically related to the ancestors of the pikes, the cyprinodonts and the mullets—related to,

but less specialized than the last. I do not look upon them as aberrant." My studies fully confirm the central position given to Menidia, for the nervous system exhibits in many of its most fundamental features the characters of a generalized type; nevertheless it is in many minor respects very highly specialized, these modifications in the nervous system being largely correlated with the exaggerated development of the organs of special sense, notably the eye and the ear.

I have studied both of these species somewhat and, so far as I know, there is no essential difference between them. The figures and descriptions are all based upon sections of Menidia gracilis. Probably they apply to both equally well.

The most essential condition of success in researches on the nerve components is the preparation of perfect serial sections. The series must be unbroken and the medullary sheaths must be perfectly preserved, for it is upon the characters of the latter that we must depend very largely to distinguish the components in the peripheral courses of the nerves. The animal chosen should be small enough to be cut whole or nearly so and should be adult or practically full grown in order that the medullary sheaths may be fully laid down. The latter point is not necessary, but it is certainly a great advantage. I have found, as many before me have done, that the teleostean tissues are refractory to a surprising degree; standard methods which work perfectly with amphibian or selachian material fail utterly here. Different species of teleosts, moreover, vary somewhat in their reactions to stains.

The necessity for very thorough decalcification of course complicates the problem greatly. In general,

rapid decalcifying solutions, and especially those containing nitric acid, are useless. So also solutions containing picric acid had to be discarded, though several, such as picro-formalin and especially picro-acetic-formalin, prove to be excellent fixers for general tissues; yet none of them preserve the medullary sheaths properly. The attempt was made to blacken the nerve sheaths with osmic acid during the process of decalcification. First Flemming's stronger fluid was tried. If applied for two or three weeks, decalcification is complete and the tissue, though very brittle, is in perfect histological condition; but the nerve fibres usually refuse to precipitate the metal and appear as white cords in the generally blackened tissues.

Hermann's fluid, if applied for several weeks, decalcifies and at the same time blackens the medullary sheaths more or less; but it does not leave the tissue in so favorable histological condition as the Flemming. Indeed, the specimens so prepared were so brittle that it proved impossible to get sufficiently continuous sections for plotting and this method was finally discarded in favor of a modified Weigert method. Nevertheless, several series which were mounted directly after fixation in Hermann's fluid, without further staining, have proved of the greatest service in controlling the others, as the sections, if once obtained, give sharper and clearer pictures of the properties of the nerve sheaths than can be secured in any other way. This applies especially to the peripheral courses of the nerves; the penetrating power of the fluid is so slight that it is not of much value centrally.

Upwards of a hundred modifications of the Weigert method were tried, for the most part with unsatisfactory results; nevertheless some of these methods are very

excellent and have given me the preparations upon which most of the work has been done. These experiments I have fully reported upon in a previous paper ('98), and I give here simply the one method which was employed in the preparation of the series upon which the plots and most of the descriptions and other figures are based. The general appearance of these sections is well shown by Figures 1 and 2, drawn for me by Mr. Veenfliet.

After fixation for two or three weeks in Flemming's stronger fluid, frequently renewed, and paraffin embedding, serial sections were cut 15 micra in thickness. Medium-sized adults were chosen, the head severed from the body behind the pectoral fins and mounted entire. To ensure the proper permeation of the paraffin it is well during the embedding to aspirate the specimen under the air pump or with a syringe and thus remove from the cavities of the body the gases evolved in the decalcification.

The sections may be fixed to the slides with Mayer's albumen, first floating them out on warm water, if necessary, to remove all wrinkles. After passing them out of the absolute alcohol one may flow over them a very thin solution of celloidin. Drain quickly, and allow to set before passing down into the lower grades of alcohol. In this way is averted the danger of the sections becoming loosened in the subsequent manipulations. From water the sections pass into a mordant composed of saturated solution of copper acetate, two volumes; water one volume. Here they remain six hours or longer at ordinary summer temperature, after which they are thoroughly rinsed in water and then treated with Weigert's hæmatoxylin for four hours or longer. Decolorize in Weigert's decolorizer, wash thoroughly (one hour or longer, in running water) and mount in the usual manner.

Section 2.—The Lateral Line Canals and their Organs.

The problems of the relations and significance of the sensory nerve components are so intimately bound up with those of the cutaneous sense organs innervated by them that an account of these organs must naturally precede the discussion of the nerves themselves. I have prepared in this section as complete an account as possible with the material at hand of the structure of the special cutaneous sense organs belonging to the lateral line system of the adult Menidia. There is urgent demand for very careful study of the comparative embryology of the various types of lateral line organs and terminal buds. The data thus far furnished by the embryologists are very "suggestive," but what they suggest must for the most part remain a matter of dispute so long as the ultimate fate of their so-called sense organs remains unknown.

I.—The Lateral Lines.

There is probably no teleostean character which is more variable in its details than the lateral line canals. In Menidia we find the system so developed as to conform very nearly to what is usually regarded as the typical form, such as is given by Allis in his diagram ('89, Plate XLII) of Amia one month old or by Cole of the adult cod ('98a, Fig. 2). If we compare with these diagrams my reconstruction of the adult Menidia on Figs. 3 and 5, the resemblance is close, the chief difference being the failure in the case of Menidia of the canals to close over the rows of lateral line organs throughout their entire extent. Thus the rows of organs are present in their typical relations, but the canals are in places interrupted.

The acustico-lateral system as a whole has been isolated from the other nerve systems and plotted upon Fig. 5, the canals being colored to correspond with their nerve supply and the limits of the related bones being indicated. This plot is drawn off from the more detailed plot on Fig. 3 and with the exception of the naked mandibular organ, *man. c.*, and the two opercular organs, *o.1.* and *o.2.*, was all drawn from a single specimen, *i. e.*, it is not a schematic or type sketch, but represents actual conditions, individual variations not being taken into account. Comparison with many other specimens shows that such variations occur, but that they are of a relatively trivial nature so that this outline may be taken as typical for the species.

In the more detailed plot, Fig. 3, the projection of the canals and their pores is represented by the green outline. The lateral line organs, like their nerves, are colored brown, those organs which are contained in canals being represented by brown rings, naked lateral line organs by brown discs. Cutaneous organs belonging to the communis system are represented by red discs, their nerves also being colored red.

In naming the lateral lines of the head I have used the following terms: The continuation of the lateral line of the trunk is termed the main line of the head up to the point where it divides behind the eye. From this point forward the line above the eye is called the supra-orbital, the line below the eye the infra-orbital. The canal of the operculo-mandibular line is interrupted between the preopercular and the articular bones. The portion caudad of this point is called the opercular line, the portion cephalad the mandibular. The incomplete cross-commissure in the extra-scapular bone is termed the occipital commissure.

The canals are also named in accordance with the bones containing them. Thus, the main canal has extra-scapular and squamosal portions, the supra-orbital canal has frontal, and nasal portions, the infra-orbital has post-orbital and lachrymal portions, etc.

1.—The Lateral Line of the Trunk.

The family Atherinidæ is characterized in the systematic works as lacking the lateral line. In Menidia the lateral line canal is absent on the trunk, but is represented by a row of very small lateral line organs innervated by the r. lateralis vagi, one for each segment of the body. Whether they extend the whole length of the body I have not ascertained. They can be followed back from the head by surface examination for only a short distance, one on each scale. But the markings on the scales of the lateral line series, *i. e.*, the groove in the centre of the scale in which the organ lies, can be recognized about half-way back to the tail. Probably minute lateral line organs extend still farther caudad.

Merkel ('80) enumerates a number of teleosts which have the lateral line developed in various degrees and there are numerous other accounts in the literature of the absence of the lateral line canals, the lines being represented by rows of naked organs. For example, Leydig ('94, p. 30) states that in Leucaspius delineatus for the caudal sixth of the body the lateral line canal fails, but the lateral line nerve continues and supplies a series of naked organs lying in a groove. The tendency for the canal to fail to appear in the trunk is doubtless to be correlated with the fact that in the ontogeny the trunk canal closes later than the head canals (Allis, '89, Leydig, '94).

2.—*The Lines of the Head.*

The lateral line canal system of the head, like that of the trunk, shows evidence of reduction or arrested development. The primary lines of the head are present in very nearly the typical form, though in part they are represented by lines of naked organs like those of the trunk line. The canals and their organs bear, so far as ascertained, the typical relations to the bones, as described by Allis for Amia ('89) and Cole for Gadus ('98a) and pores are present between each pair of organs, the only exception being the space between the first organ of the trunk canal and the organ of the occipital canal, between which there is no pore. The pores are always short and simple, never showing the dendritic arrangement found in some other fishes. The lateral line organs of the head, whether contained in canals or not, are always supplied by the acustico-lateral system of nerves, and these nerves never supply any other organs.

This is a point which can be determined with certainty in nearly every case by reason of the great size of the lateral line fibres, as compared with those of any other sensory system. These fibres, when bound up in a common sheath with others, usually segregate themselves, so that they occupy a definite portion of the cross-section of the nerve, and this renders their separation still more easy. The most serious difficulties were encountered in distinguishing certain naked organs of the lateral line system, which correspond probably with the pit-lines of Amia, from terminal buds. These organs are sometimes innervated by fibres which are intermediate in character between those of the lateralis and communis systems, being very densely myelinated and hence staining very

dark, like the former fibres, and yet of small size. They are not, however, so small as the ordinary communis fibres.

3.—The Extra-scapular Canal.

i.—The Post-Occipital Main Canal.—Just cephalad of the pectoral fin the canal system of the head appears, as the direct continuation of the lateral line of the body. As we pass cephalad this canal is first a dermal groove which very soon closes to form a narrow canal in the dermis and the latter at once sinks down into a groove in the extra-scapular bone. At the level of the last sense organ of the head (715 on the plots) the bony groove has become a canal. In this osseous canal the membranous canal expands to the normal width, which is maintained with tolerable uniformity throughout the entire canal system of the head.

The sense organ above referred to (*m. 3* of Fig. 5) is the only one of the main line behind the occipital commissure which is enclosed in a canal. It is supplied by the first ramus of the r. lateralis vagi (*r. l. 1.*), excluding the r. supra-temporalis. This organ might be regarded either as the first one of the trunk series or the last one of the head series.

ii.—The Occipital Commissure.—The incomplete occipital commissure branches off from the main canal at an acute angle and extends dorsad and cephalad only about one mm. before it opens out and disappears. A single elongated canal organ is found in this canal (Fig. 1, *m. 4*). It lies very near the origin of the canal and is supplied by a branch of the r. supra-temporalis vagi. No naked buds could be found in the skin along the line continuing the course of the canal, as is the case behind the lateral line canal.

After its separation from the main canal the occipital commissure remains in the extra-scapular bone. The main canal, however, soon leaves this bone, lies close under the skin and immediately opens out for a short dis-

tance (660–675) as a wide shallow pore. There are no pores in the main canal caudad of this point, nor in the occipital commissure.

4.—The Squamosal Canal.

Cephalad of this point the main canal sinks down again and is enclosed in the squamosal (pterotic) bone and in this portion of the canal is included the single sense organ of the main line lying between the occipital commissure and the opercular canal (*m. 2*). This organ is innervated from the r. supra-temporalis vagi. Farther cephalad the canal comes to lie in a deep narrow groove in the bone up to the point of union with the opercular canal. At this point a rather long narrow pore is found, directed caudad and ventrad close under the skin.

After giving off the opercular canal, the main canal (550) sinks again into a deep groove in the squamosal bone and there is lodged the single canal organ of the main canal between the opercular and the infra-orbital canals (*m. 1*). It lies nearer to the former than to the latter and is innervated by the r. oticus. The canal runs in this groove very nearly to the point where the infra-orbital canal diverges from the supra-orbital, a tongue of the squamosal bone running forward to accommodate it. Here the canal communicates with the surface by means of a pore.

The operculo-mandibular canal is separated below the eye into its two portions, opercular and mandibular, which will be separately described, the canal organs and pores of each being numbered from before backward.

5.—The Opercular Canal.

The opercular canal lies for almost its entire length in a groove in the caudal and ventral faces of the preopercular bone, which ventrally extends forward to the mandible. The canal has a vertical and a horizontal limb of nearly equal length and contains seven canal organs, three in the vertical and four in the horizontal limb, the former

of which are innervated from the truncus hyomandibularis, the latter from the r. mandibularis externus VII. The groove in the preopercular bone is much deeper in the vertical than in the horizontal limb and for a small part of its course the bone entirely encloses the canal, forming an osseous canal.

There is no pore between the seventh opercular canal organ and the main canal. The pore between the sixth and seventh organs (*o. p. 6*, Fig. 5) passes dorso-caudad, the fifth and fourth pores pass ventro-caudad, and are rather longer than the sixth. Between the third and fourth organs the canal again lies close to the surface and the pore is a mere break in the outer wall of the canal with no considerable tube. The first and second pores are similar to the third. For an illustration of a typical arrangement of these pores see Fig. 27.

The opercular canal extends cephalad to 290, always in the groove of the preopercular bone except at the extreme cephalic end. After leaving this bone the canal almost immediately comes to the surface, opens out and disappears.

6.—*The Mandibular Canal.*

The mandibular canal appears less than one millimetre in front of the cephalic end of the opercular canal (235) and its course is a direct continuation of that of the opercular canal. It immediately sinks down into a groove in the articular bone by which it soon becomes entirely enclosed. There are five organs in the mandibular canal, of which the fifth lies in the articular bone, the others in the dentary. They are all innervated from the r. mandibularis externus VII.

About midway between the fifth and fourth canal organs is the fourth mandibular pore. The third pore lies nearer the third organ and the second pore nearer the second organ, while the first pore arises almost directly over the first organ. The mandibular canal ends in a pore at the extreme tip of the mandible without communicating with the canal of the opposite side.

7.—*The Infra-Orbital Canal.*

This canal passes ventrad and slightly caudad and is enclosed at once by the most dorsal of the post-orbital bones. Its course is very short, for as soon as it has passed this bone it opens out and disappears. This bone is rather massive, much more so than any of the other bones of the orbital ring.

There are no pores in this short section of the canal and but one sense organ. The infra-orbital canal is absent from the most caudal edge of the orbit to a point a little beyond its ventral edge, where it resumes. This short portion of the infra-orbital canal we shall call the post-orbital section, the pre-orbital portion, the lachrymal section.

It is interesting to note that the entire orbital ring of bones in Menidia is very much reduced. In view of the fact that the lateral line organs of the head normally lie in bony canals and that, even if the canal is not entirely enclosed in bone, there is a tendency to form bridges of bone over the organs themselves (as, for example, in the supra-orbital canal), it would seem reasonable to conclude that the absence of that portion of the infra-orbital canal which is normally enclosed in the bones of the orbital ring is correlated with the reduction of those bones.

The portion of the infra-orbital line which lacks the canal is represented by a series of naked sense organs which in shape resemble the terminal buds more than they do the canal organs. Those near the open ends of the canals are larger than those in the middle of the naked series. They are all innervated, however, by the coarse fibres of the r. buccalis belonging to the acustico-lateralis system.

There are fifteen sense organs in the infra-orbital series, the first five in the lachrymal section of the canal, nine naked organs in the ventro-caudal quadrant, and one canal organ in the short post-orbital section. The latter and the last two naked organs are innervated by the r. oticus, all of the other organs by the r. buccalis.

Upon the reappearance of the canal in front of the orbit (260), it sinks down into a deep groove in the lachrymal bone and at the level of the fifth infra-orbital sense organ the bone arches up so as to entirely enclose the canal. Between the fifth and fourth canal organs there is no pore. Arising almost directly over the fourth organ is the fourth pore of this series, which is directed cephalad. There is a similar one over the third organ. The second pore lies ventrally of the second organ and the first pore dorsally of the same organ and a little farther removed. Thus the number of pores corresponds to the number of spaces between the organs, though they are somewhat displaced from their normal positions.

The infra-orbital canal terminates, after rising to the top of the head and bending slightly caudad, in a pore which lies near to and laterally of the posterior nasal aperture and separated only by the latter from the supra-orbital canal. It does not communicate with any other canal system.

8.—The Supra-Orbital Canal.

After separating from the infra-orbital, this canal sinks at once into a groove in the frontal bone. It contains six canal organs, all supplied by the r. ophthalmicus superficialis VII. This groove at the level of the sixth organ is roofed by a narrow bridge of bone and just cephalad of this point is a narrow pore. The fourth and fifth canal organs are close together and are roofed over by similar narrow bridges of bone. The corresponding pore (fourth) lies over the caudal portion of the fourth organ. The third pore is longer, narrow and directed dorsad and caudad. From this point forward the canal is wholly enclosed by the frontal bone as far forward as the second pore, which lies just behind the second organ. Somewhat cephalad of this point (190) the frontal bone disappears, its place as bearer of the canal being taken by the nasal, in which the first organ of this canal lies. The first pore lies just behind this organ and from this point to the end

of the canal (90) the bone entirely encloses the canal. The canal ends in a minute pore some distance from the tip of the snout dorsally of the anterior nasal aperture and does not communicate with any other canal.

II.—Accessory Lateral Line Organs.

Under the names of accessory lateral lines or pit-lines other writers have described rows of naked cutaneous sense organs found in various places on the skin of different fishes and innervated from various sources. The morphological significance of all of these structures is very obscure and can probably not be definitely settled until we have more accurate knowledge of their nerve supply and development. Unfortunately Menidia is not a favorable type for the solution of these problems, as the cutaneous sense organs are all developed much less highly than in many other fishes.

In many fishes and amphibians there have been described rows of naked organs on the trunk, the so-called accessory lateral lines. The most constant of these is the dorsal accessory lateral line, which runs parallel with the main lateral line near the mid-dorsal line. Another may run parallel with and ventrally of the main line, while a third series of organs may be distributed in various ways along the course of the main line. In some cyprinoid fishes such organs are scattered over the whole body, each scale bearing one or more. Any or all of these organs may be innervated from branches of the r. lateralis vagi or the dorsal and ventral lines may be supplied, as in the gadoids, by branches of the r. lateralis accessorius (r. lateralis trigemini, or superficial lateral line nerve, of authors). This latter nerve I have shown to belong to the communis system. In almost all cases when these

accessory organs on the trunk are supplied by the r. lateralis vagi that nerve is described as receiving anastomosing fibres from either the vagus, glossopharyngeus or facialis which are apparently of communis origin. These organs are commonly considered to belong to the lateral line system and to be similar to the pit-lines described by Allis on the head of Amia; but before such a conclusion can be safely accepted it must be definitely determined that their nerves are of lateralis and not communis origin.

These accessory lateral line organs of the trunk are represented in Menidia by a few naked sense buds along the course of the r. lateralis accessorius (see figures 3 and 4). They are innervated from anastomosing branches from the r. lateralis vagi. The fibres are of small or medium calibre and are, I think, derived from the lobus vagi by way of the anastomosis from the IX root to the lateralis root. A row of similar organs is found farther caudad in very young specimens lying directly over the course of the r. lateralis accessorius. Their nerves could not be traced, though there can be no doubt that they are derived from the nerve last mentioned, especially as such a condition has been described for several other fishes.

I have no sections running through the trunk of the adult, but surface examination with a lens is sufficient to demonstrate that this dorsal series of naked organs persists to adult life. Specimens preserved in ten per cent formalin show the organs better than alcoholic material. One or two organs are found above the operculum near the one at 620 on the plots, but never very many. The dorsal body line is represented by an irregular series of organs close to the median line and directly over the position occupied by the accessory lateral nerve. These organs are not numerous and do not occur on each scale as we pass caudad. They are most conspicious in the region of the dorsal fin, which lies rather far caudad, but even here they are not regularly arranged. Between the dorsal fins and the head only a very few scattered organs are found.

From the innervation of these organs I incline to regard them, like the buds on the top of the head, as belonging to the communis system. Of course it does not necessarily follow that the accessory lateral lines of Fierasfer, Amia, etc., which are innervated by branches of the r. lateralis vagi, are of the same nature. See the further discussion of the r. lateralis accessorius in Section 12.

Upon the head there are several series of naked cutaneous organs which are clearly innervated by the lateral line nerves and which I homologize with the pit-lines of Amia. See fig. 5.

One row of four large organs (*o.1* to *o.4*) follows the ventral edge of the operculum behind the fourth pore of the opercular canal. One organ (and I think usually two) lies on the dorsal surface of the operculum behind the sixth pore of the opercular canal (*o.5*). These five organs are innervated from the r. opercularis superficialis VII and obviously from the coarse-fibred lateralis component. Another similar organ (*o.6*) lies in front of the opercular canal at the level of the fifth pore and is supplied by the first branch of the r. mandibularis VII.

Along the course of the horizontal limb of the opercular canal and just external to the canal are three groups of similar, but smaller, organs innervated respectively by the first, fourth and sixth branches of the r. mandibularis VII. A group of three similar organs lies along the course of the mandibular canal and is supplied by the eighth branch of the r. mandibularis VII. A single organ on each side (*man. c.*) lies mesally of the cephalic end of the mandibular canal, the two forming a mandibular commissural line.

There are four organs in the nasal region which are supplied by the r. buccalis (*a. d. e. f.*) which appear also to form a similar pit-line, making, together with a line from the opposite side, a maxillary commissure. About the anterior nasal aperture there are five organs, two of which (*b. c.*) are supplied by the r. buccalis, and three (*g. h. i.*) are supplied by the r. ophthalmicus superficialis. They are of uncertain nature. I have provisionally

reckoned them all among the pit-organs. The evidence for this is chiefly comparative, as the nerve supply is ambiguous; see the account of their nerves in Section 7.

The morphology and homologies of all of these pit-organs are further discussed in the pages immediately following and under the head of their nerves in Section 7.

It should be noted that the "pit-organs" of Menidia are not situated in pits, as in ganoids and as in the cod (Cole, '98a). They are strictly naked papillæ projecting above the surface of the skin. Cole (p. 187 ff.) argues with great force that pit-organs, visicles of Savi, ampullæ and lateral line canals, represent a progressively advancing series in the differentiation of these organs. This seems probable, and in that case the pit-organs of Menidia stand nearer the primitive condition than do those of the ganoids.

III.—Comparative.

In all there are thirty-seven organs belonging to the lateral line system proper, exclusive of pit-organs, on each side of the head of Menidia. In Amia Allis' enumeration shows that there are forty-seven (forty, not counting those in the extra-scapular and the other more posterior bones, '89, p. 499), besides the "spiracular organ." To get the former number he enumerates the one organ found in the supra-scapular bone and the two in the supra-clavicular as belonging to the head, thus including three organs behind the extra-scapular bone, while I have included none behind the latter bone, since neither of the other bones comes into relation with the lateral line. In Gadus Cole ('98a) describes thirty-two canal organs in the head. In the distribution and innervation of these organs, Menidia exhibits a striking general similarity to both of these types, though the parallelism is not

exact. Cole gives (pp. 179–185) a brief but very excellent summary of the more interesting variations of the lateral line canals of different kinds of fishes.

The numerous writers who have described the lateral canals of the fishes have, as a rule, until recently, devoted no considerable attention to their nerve supply so that the correlation of their results is in many cases a matter of difficulty. Pollard first clearly demonstrated in teleosts the independence of the lateral line system of nerves; but both his work and that of Collinge leave much to be desired to complete our knowledge of the innervation of the cutaneous sense organs in the teleosts and especially in the siluroids, which are of special importance in this connection. Pending further studies here, it will not be necessary to examine their work in detail. The conditions in Lophius, however, as worked out by Guitel ('91), shed some light upon our findings and will be here reviewed.

The diagram, fig. 6, adapted from Guitel, exhibits the topographical relations of the lateral lines and their innervation. In spite of the fact that this system is very highly developed in the head, there are no canals in Lophius. The organs lie in grooves or pits and are overlapped by the peculiar dermal fringes so characteristic of the Pediculati. The absence of the canals is probably to be correlated with the fact that the skeleton is remarkably loosely aggregated. Indeed, the older writers classed this among the cartilaginous fishes. Guitel describes several of the head lines as innervated from the trigeminus. Of course in the light of present knowledge we must relegate these branches to the facialis, and with this correction the homologies of his lines are clear.

In Lophius there is but one lateral line on the trunk,

which is supplied by the r. lateralis vagi. There are three dorsal branches of the lateral line nerve, of which it is clear from the description that only one contains proper lateralis fibres. The first of these is an opercular nerve which goes to the skin of the operculum, the "superficial opercular," and evidently corresponds to my branches *cut. X. 2* and *cut. X. 3* (see Section 5, VIII). It also sends a branch, the "opercularis profundus," which anastomoses with the r. opercularis profundus VII and supplies the lining of the branchiostegal rays and which evidently corresponds to my branch *n. op. X.* The second dorsal branch is motor and apparently corresponds in part to my branch for the trapezius muscle (*XI*). The third dorsal branch is the proper supra-temporal nerve. It supplies the lines (see fig. 6) *p-o*, *p-b*, *o-b* and *b-l*. These lines evidently represent the supra-temporal commissure and certain of the pit-lines found in Amia.

The lines *b-d*, *d-e*, *d-k*, *e-h*, *e-f*, *f-g and f-m* are all innervated by the r. mandibularis externus facialis, and correspond to the operculo-mandibular lines of Menidia. In Lophius there are some anastomoses of these nerves with the r. mandibularis trigemini, but there is no evidence that any of the lateralis fibres come from the latter source. The r. mandibularis externus VII is not, as in Menidia, a single nerve, but is broken up into several independent nerves. Thus, the lines *b-d*, *d-e*, *d-k* and *e-h* are supplied by a single branch which Guitel calls the r. opercularis superficialis VII, and which corresponds to that nerve in Menidia plus a portion of the main r. mandibularis externus VII. The line *b-d-e-f-m* corresponds to the operculo-mandibular line. The other lines of this series I think are to be compared with pit-lines of some other forms. Thus, the line *e-h* corresponds in

position and innervation with the pit-line *o.1* to *o.4* in Menidia, and the line *d-k* to the organ *o.5* on the dorsal surface of the operculum of Menidia. In Batrachus (Clapp, '99) there are lines in these positions like those of Lophius. These lines also bear some resemblance to the gular and jugular lines of Chlamydoselachus, and the organ *o.6* to the angular line of the same fish (Garman, '88). The line *d-k* apparently also corresponds to the posterior limb of the hyomandibular canal of selachians (Ewart, '93) and Chimæra (Cole, '96a), and in the latter cases there are groups of ampullæ in the same region, which probably also belong in the same category. In Amia (Allis, '89) there are pit-lines supplied by similar branches of the hyomandibular nerve, but these lines lie cephalad of the opercular canal instead of caudad of it.

The line *f-g* of Lophius lies in the same position as an area of thickened epidermis in Menidia under the eye between the infra-orbital and opercular and mandibular canals, an area containing many pit-organs in Gadus.

The line *q-c-n-g-d* of Lophius would at first sight be compared directly with the infra-orbital line of Menidia. Yet, in spite of the fact that both lines are innervated by the r. buccalis, the homology is, I think, only incomplete, as shown by the peculiar course of the nerve in Lophius and a comparison with Batrachus (Clapp, '99). In the latter case there is a small (and obsolescent?) infra-orbital line and *in addition* a "maxillary line" which runs caudad from near the cephalic end of the infra-orbital, laterally of the latter, but does not reach the opercular line. This latter line is more highly developed in Lophius, while the caudal portion of the infra-orbital line, which should connect with the cephalic end of the line *l-b*, has disappeared. That the line *n-g-d* of Lophius has grown

back from in front of the eye is suggested by the course of its nerve, which first runs forward cephalad of the eye and then sends recurrent twigs to supply the line back to the point where it joins the opercular line. The line *o-c* represents the supra-orbital canal of Menidia. It is probable that the organs belonging to the r. oticus are also included here, for the last organs of this line are innervated by a separate branch of the r. ophthalmicus superficialis VII. Upon comparing the diagram of Lophius with Allis' diagram of the lateral line system of Amia, it is suggested that the line *o-p* is the occipital commissure and its pit-line of Amia, the line *o-b* is the middle pit-line, while the anterior pit-line is represented in the caudal part of the line *o-c*. This last supposition would explain the fact that the supra-orbital line does not join the main line *l-b*, but the others farther dorsad.

Material has been collected and some fragmentary observations have been made upon the development of the lateral lines in Menidia, but these results are as yet too incomplete to yield much of value. In very young specimens about 1 cm. long the cutaneous sense organs were plotted and all of the lateral line organs were found in essentially the same relations as in the adult save that no canals are developed. The number and arrangement of the naked lateral line organs is the same as in the adult. In specimens a few mm. longer the canals have begun to appear and when 2½ cm. long the canals have been completed very nearly as in the adult. The pores are in all cases wider at this age than in the adult and the main canal is interrupted for the entire distance between the point of union of the supra- and infra-orbital canals and the organ lying next caudad. Only four organs are contained in the lachrymal segment of the infra-orbital canal,

instead of five as in the adult, and the mandibular canal has not been closed so as to cover the first organ of that line.

Is the simplicity of the lateral line system in Menidia as compared with many other fishes, especially the lower fishes, to be regarded as primitive simplicity or as the result of degeneration? Cole would say the former, for he argues ('98, p. 245) that the naked condition of the sense organs is always the primitive and that in the decline of the system these organs are lost before the canals. But how about the Amphibia in which the system is fluctuating on the verge of extinction and yet no canals are present, only naked organs? On the whole I incline to regard the condition in Menidia as reduced rather than primitive.

The various recent attempts to show that a part of the lateral line system is innervated by branches of the trigeminus, such as that of Collinge ('95), doubtless rest either upon faulty observation or loose definition. Perhaps the clearest of these cases is that of Chimæra (Cole, '96a) in which two organs of the supra-orbital line are innervated from the profundus; but Cole himself feels confident that a microscopical examination would show that the nerve in question is really a twig of the lateralis system which is detached from the r. ophthalmicus superficialis and secondarily joined to the profundus—a question easily answered by a determination of the central connection of these fibres, whether in the lateralis or the Gasserian ganglion.

Another case difficult of interpretation is given by Miss Platt ('96, p. 530), for Necturus. "I have traced the nerve twigs to each one of the terminal clusters of organs on the infra-orbital line, and find that four of the organs, which I have marked in the reconstruction, are supplied

by nerve twigs composed in equal parts of fibres coming from the buccalis facialis and from the ophthalmicus profundus. These fibres unite in a common twig that goes directly to the heart of the sense organ." This observation is of the highest importance and should be verified if possible upon adult material. Without questioning the accuracy of Miss Platt's observation, a consideration of her description and figures suggest certain cases which I have observed in the case of Menidia, where a general cutaneous twig goes out with the lateralis twig for a sense organ and just before that organ is reached turns abruptly to one side to supply the skin adjacent to or overlying the canal organ. In an undifferentiated embryonic tissue these smaller cutaneous fibres might be overlooked, or they might not at the stage studied have attained their ultimate growth toward the skin.

IV.—Summary of the Lateral Lines.

The lateral line system of Menidia is not highly developed, but presents very nearly the typical arrangement of lines. The canal is wanting in the trunk and in a portion of the infra-orbital line, though these lines are represented by rows of naked lateral line organs. The organs of the lateral lines, whether contained in canals or not, are always innervated by the acustico-lateral system of nerves. The canals are related to the bones of the head in the typical manner, as described by Allis for Amia. A simple pore is normally found between each pair of organs. The lateral lines are accompanied in some cases by rows of naked organs which are also innervated from the acustico-lateral system of nerves and which appear to correspond with the pit-lines of Amia. These pit-lines are in some other fishes represented by true lateral lines.

Section 3.—The Central Relations of the Cranial Components.

Before proceeding with the description of the nerves it will be profitable to discuss the terminal relations within the brain of some of the components of the cranial nerves and their probable relation to the spinal components. The motor nuclei can best be described in connection with the several cranial nerve roots to which they give origin; they will, therefore, in this section receive only a brief general treatment. The sensory terminal nuclei are, however, grouped into systems each of which is related to several of the cranial nerves as ordinarily enumerated. A preliminary description of each of these systems, taken as a whole, will assist in the ultimate analysis of the cranial roots, ganglia and rami which are related to them.

I.—The General Cutaneous System.

The general cutaneous nerves supply general sensation to the skin without specialized sensory end-organs. Nerves from lateral line organs and from terminal buds are excluded from this system. These nerves correspond in every respect, however, with the cutaneous fibres of the spinal nerves and are serially homologous with them.

In Menidia this system is represented in the V and X cranial nerves only. Such fibres are said to occur in the IX nerve of other forms—Selachians, Ewart ('89 and '92), Chimæra, Cole ('96, p. 664), and Acipenser, Johnston ('98, p. 585).

By far the larger part of these fibres arise from the Gasserian ganglion, and these make up the whole of the sensory trigeminus root. They are so intimately joined to the motor V fibres that within the brain it is not possible to separate the two components perfectly. (Fig. 20).

Most of these fibres turn caudad immediately upon entering the brain and constitute the spinal V tract. A large bundle accompanies the motor root nearly to the median line and constitutes the "deep portion of the descending Vth" of Johnston ('98). Most of these fibres, both sensory and motor, pass at once to the opposite side through the commissura accessoria, but some of the motor fibres terminate in, or more strictly, arise from, the motor V nucleus and the fasciculus longitudinalis dorsalis of the same side. The sensory fibres of this bundle probably also, in part, cross to the opposite side, though they could not be separately followed. Some of them appear to end in a compact nucleus of very small cells lying very near to the motor V nucleus and a little farther caudad. This I take to be the "chief sensory nucleus" of the trigeminus. It should be stated, however, that my knowledge of this nucleus and its connections is not as precise as that of the other roots described. No considerable number of trigeminal fibres turn cephalad from the origin of the nerve. The nucleus lying under the cerebellum to which Johnston traced sensory trigeminal fibres in Acipenser was found, but no fibres were traced to it, nor were the descending cerebellar fibres described by him and by Goronowitsch discovered. No Golgi preparations were made and I cannot deny the presence of such fibres in relatively small numbers, as this region has not been exhaustively studied.

The spinal V tract runs back very close to the lateral wall of the oblongata and ventrally of all of the sensory VII and of the VIII roots, but dorsally of the motor VII (Fig. 19) and motor IX (Fig. 18) roots. The sensory IX fibres emerge dorsally of it, the X fibres both dorsally and ventrally. As the lobus vagi increases in size it crowds

the spinal V tract laterally and dorsally until, at the level of the caudal vagus roots, it occupies the extreme dorso-lateral angle of the oblongata, where it forms a projecting cord or band. This relation is maintained back to the level of the exit of the first spinal nerve, where the spinal V tract merges into the nucleus funiculi and the dorsal cornu.

Between the cephalic and caudal vagus roots the spinal V tracts receives a small bundle of fibres from the jugular ganglion of the vagus.

Haller ('96, p. 64), describes for Salmo, Perca and Anguilla, and figures in the latter case a large tract running from the lobus vagi adjacent to the ventricle to emerge with the trigeminus root. He also figures a smaller root running from the lobus vagi into the VII nerve and says that the chief trigeminus nucleus is directly continuous and serially homologous with the lobus vagi.

Haller's account of the V and VII roots in the eel appeared to me so remarkable that I have myself examined the matter. From a study of both transverse and longitudinal sections of the brain of Anguilla chrysypa Raf. it appears that the V and VII roots of the eel are typical, as compared with Menidia, and that the differences between Haller's account and mine arise from the fact that he has wrongly identified several of the roots.

In Anguilla, the lateralis system is developed about as in Menidia; the general cutaneous system is the same in plan, but enormously developed, the spinal V tract being especially enlarged; the communis system is much reduced. The lobus vagi and its roots are small, doubtless correlated with the reduction of the gills. The communis root of the facial is, however, very large. It runs, as in Menidia, directly to the ventricular wall and turns

caudad, as the fasciculus communis, into an elongated lobus IX + VII. The latter receives the sensory IX root and is continuous caudad with the lobus vagi.

Now, the root which Haller calls trigeminus (Plate IV, fig. 26) obviously contains, besides the sensory and motor V, the communis root of the VII and probably also the dorsal lateralis root of the VII, while his root marked *ac.+fac.* contains the ventral lateralis root of VII, the motor VII and perhaps VIII fibres. The portion of the latter complex which he marks *fac.* is not a sensory facialis root terminating in the cephalic end of the lobus vagi, as Haller supposes, but the motor VII root, which passes out from its nucleus via the fasciculus longitudinalis dorsalis, exactly as in Menidia.

The root which Haller calls the "obere innere Ramus ascendens n. trigemini (*r. a. tr. sup.*)" and considers as a root from the cephalic end of the lobus vagi is the communis root of the facialis, *i. e.*, the fasciculus communis. Its terminal nucleus, the lobus VII+IX, is continuous with the lobus vagi, and it is not true that the latter "is, accordingly, not sharply defined forward, but is continued without interruption into the upper or sensory trigeminus" (p. 64); for none of the centres in question have anything whatever to do with the trigeminus.

Haller supports the serial homology of the lobes from which the V, IX and X nerves arise by citation of the case of Lota vulgaris, but upon comparing the figure given with a similar figure of the same species by Gorono-witsch in the same Festschrift it is difficult to see any resemblance between the two either in the number and forms of the lobes or of the nerve roots, and little reliance can be placed on any of this evidence until this species is re-examined.

Johnston ('98, p. 594) describes the sensory trigeminus in the sturgeon as arising from the spinal V tract and from the tuberculum acusticum. The latter fibres apparently correspond with those to the chief sensory nucleus of Menidia and other forms. There is no evidence that they terminate in the tuberculum acusticum proper.

The "system *y*" of Goronowitsch ('88 and '96) in Acipenser corresponds to the spinal V tract and the secondary vago-trigeminus tract of teleosts and the fibres which enter the system *y* in Acipenser from the lobus vagi ('96, p. 9) and in Lota from the lobi vago-glossopharyngeo-facialis ('96, p. 21) are obviously the secondary and not the root fibres. In Menidia the secondary vagus bundle (of Mayser) follows the inner and ventral side of the spinal V tract, but can always be clearly distinguished from it (Figs. 17, 18, 19, *Sec. X*). The motor IX and VII roots run between a portion of the secondary vagus bundle and the spinal V. The secondary VIII bundle lies dorsal and somewhat removed from the spinal V (Figs. 18, 19, *Sec. VIII*).

II.—The Acustico-lateral System.

This system includes the nerves which supply the lateral line organs and kindred structures, viz.: the r. lateralis vagi, the VIII nerve and the two lateralis roots of the facialis. Their nerves terminate together in the tuberculum acusticum for the most part, and their fibres are very intimately intermingled.

In the middle portion of the cerebellar peduncles the outer, or molecular, layer disappears laterally, the cerebellum being bounded on these aspects by the fibrous and granular layers only. These layers are continued caudad into the tuberculum acusticum of the oblongata and

receive from the caudal edge of the cerebellum a cap of the molecular layer, the cerebellar crest. Still farther caudad the two cerebellar crests, immediately after their separation from the cerebellum, fuse in the middle line dorsally of the fourth ventricle (Fig. 18). The fusion is substantial, involving the molecular layer, the now rudimentary granular layer and the underlying fibres of the tuberculum acusticum proper. The molecular layer caps the tuberculum for its entire length, *i. e.*, caudad beyond the cephalic end of the lobus vagi and nearly to the caudal tip of the cerebellum. Its extreme caudal tip is shown in Fig. 17.

The cerebellum we know to be related to the secondary tracts of all of the sensory nerves. It is said by several authorities to receive direct root fibres from the trigeminus; but its direct relations to the roots of the acusticolateral nerves is strikingly characteristic of this system.

Several large bundles of root fibres from the VIII nerve (Fig. 19, *cb. VIII*) can easily be followed in the transections up into the cerebellum near its cephalic end. Probably other similar bundles run into the cerebellum from the three lateral line roots, as described by Johnston ('98).

The VIII nerve and probably also the three lateral line roots send root fibres caudad, thus constituting the spinal VIII tract. These fibres form two close round bundles lying at the periphery of the oblongata. The sensory root of the vagus emerges just dorsally of them (Fig. 17, *sp. VIII*). Immediately caudad of the level here figured (640) they turn ventrad, forming external arcuate fibres to cross in the extreme ventral portion of the raphe. This decussation occupies the extreme ventral surface of the brain for almost the entire extent of the region of the lobus vagi.

Goronowitsch ('96) describes and figures this tract in Lota under the name of "ascending Trigeminus II" and finds it derived from the lateral line VII (his Trig. II, dorsalis) and from the VIII. It appears to be homologous with the spinal VIII of human anatomy. It is doubtful whether it is completely homologous, if at all, with the spinal VIII of Acipenser, as described by Johnston ('98). The latter tract appears from the description to be made up largely of secondary fibres from the tuberculum acusticum, and not, as here, and as in human anatomy, of direct root fibres. Moreover, Johnston's tract runs back closely joined to the spinal V tract and mesally of it, to terminate in a dorsal nucleus lying mesally of the nucleus funiculi. The tract to which Johnston gives the name spinal VIII is apparently the tract which I term the secondary VIII bundle (Figs. 18 and 19). In Menidia this tract is apparently composed mainly of ascending fibres, though it may contain descending fibres, such as Johnston describes, also.

The secondary fibres arising in the tuberculum acusticum for the most part cross in the commissura accessoria Mauthneri. Some, however, enter a secondary VIII bundle on the same side. The tract to which I have given this name (figs. 18 and 19, *Sec. VIII*) is composed mainly of uncrossed fibres, but partly, I think, of crossed fibres. It passes into the cerebellum. The other secondary fibres from the tuberculum acusticum, after crossing in the commissura accessoria, enter the tractus bulbo-tectalis (*tr. b. t.*), and most of them, if not all, pass directly up to to the optic tectum.

III.—The Communis System.

Osborn ('88, p. 63) applies the term fasciculus communis to a tract in the amphibian oblongata because of

"its common relations to a number of the cranial nerves." In the Amphibia the fasciculus communis receives most of the root fibres belonging to what I have called the communis system. These enter with the VII, IX and X roots. The fasciculus is a tolerably uniform tract which, after receiving the root fibres above mentioned, continues into the spinal cord uninterruptedly as far as the first spinal nerve, being accompanied for almost its entire length by a strand of nerve cells which constitute a terminal nucleus for its fibres, the spinal nucleus of the fasciculus communis. This tract is the "Radix bulbospinalis Vagi et Glossopharyngei" of Edinger ('96, p. 84).

In the fishes, however, the greater part of the fibres of this system enter the oblongata through the chief vagus root and pass directly to their terminal nucleus in the lobus vagi without entering the fasciculus communis in the original sense of that term. The chief root of the vagus is in the fishes (and doubtless in the higher forms also) a complex of at least three kinds of fibres: (1) Sensory fibres from the general visceral surfaces without specialized end-organs. (2) Sensory fibres from taste buds in the mouth and from similar terminal buds variously scattered over the outer surface of the body. It is generally assumed that these two classes of buds have a common origin, as well as a common structure and innervation. They must be sharply distinguished from the neuromasts, or organs of the lateral line (nerve hillocks of Merkel), which belong to a distinct system. (3) General cutaneous fibres from the outer skin. The latter category and all motor fibres are excluded from the communis system.

In the communis system, then, there are represented two types of fibres, the general visceral and the taste bud

(and terminal bud) fibres, which appear to be quite distinct from each other functionally and may be so anatomically, though we cannot as yet effect their analysis. The pre-auditory portion of the fasciculus communis here, as in the Amphibia, is composed mainly, though probably not wholly, of fibres of the second of the classes enumerated above, and it was these fibres which Strong had primarily in mind in proposing the term "fasciculus communis system." In the tract as a whole, however, he recognized both types of fibres and also the motor component ('95, p. 182).

Now the fact that these kinds of fibres are developed in varying degrees in different animals and the fact that more or less of their terminal nuclei have sometimes been included with them under the term fasciculus communis have already occasioned considerable ambiguity as to what is meant by this term and the matter of definition becomes important. I repeat, therefore, that under the term *communis system* I include the sensory cranial nerves supplying the visceral surfaces, taste buds and terminal buds, their ganglia, root fibres, peripheral end-organs and terminal nuclei in the medulla oblongata. The term fasciculus communis I shall use in its original sense as a tract of fibres running from the seventh nerve caudad in the oblongata and receiving in different animals varying proportions of the root fibres of the communis system. Other root fibres of that system may pass to their terminal nuclei directly without entering the fasciculus communis. The fasciculus communis may contain in some part of its course viscero-motor fibres; but, if so, such fibres are not regarded as belonging to the communis system, which is wholly sensory.

In Menidia the pre-vagal portion of the fasciculus communis contains a portion of the root fibres of the VII and IX nerves, as described in the sections devoted to those nerves. I find no indication of a pre-facial fasciculus communis. From the communis root of the facialis this tract passes back to the lobus vagi as a compact round bundle lying close to the ventricle. After receiving the sensory IX nerve (Fig. 18) it begins to be surrounded by an area of "ground substance" and almost at once enters the lobus vagi in several strands.

The lobus vagi crowds the other structures of this region laterally until it occupies nearly the whole of the dorsal part of the oblongata. Upon almost the whole of its lateral face the root fibres of the vagus are received. The lobi vagi are very moderately developed as compared with some other teleosts, *e. g.*, cyprinoids, and fuse in the median line only at their caudal extremities over the tip of the fourth ventricle. The small size of the lobus vagi may be correlated with the reduction of the terminal bud system in Menidia.

The motor vagus nucleus (nucleus ambiguus) lies ventro-mesally of the lobus, just laterally of the floor of the ventricle and dorsally of the fasciculus longitudinalis dorsalis. (Fig. 17). Caudad of the exit of the motor vagus roots the lobus diminishes in size and the other dorsal structures, *i. e.*, the dorsal cornu, nucleus funiculi and spinal V tract, appear in their normal relations. In the spinal cord of these fishes, it should be noted, the dorsal horns are crowded mesally as far as possible, so that they lie up against the dorsal fissure with practically no white column intervening (Fig. 16).

The following description, though based primarily upon Weigert sections, has been controlled by the examination of a series of sections of the brain of Menidia stained by Nissl's method. The cells of the lobus vagi are minute and densely crowded in a narrow zone along the dorsal and mesal surface of the lobus close under the endyma, with but few cells in the interior of the lobe. On the

caudal face of the lobus vagi medullated fibres gather and constitute a spinal portion of the fasciculus communis; some of these cross at once to the opposite side dorsally of the fourth ventricle in small irregular bundles. Others continue caudad, where they are joined by additional fibres arising farther laterally, probably from the dorsal cornu, and just caudad of the lobus vagi they participate in the formation of a compact commissure or decussation. All of the transverse fibres above described belong to the commissura infima Halleri, which in Menidia is but feebly developed.

From this place, on each side, a close round bundle of medullated fibres extends caudad to the level of the first spinal root, where it breaks up and disappears in an area of loose reticular tissue which contains few medullated fibres and many rather small pale cells with scattered larger deeply stained multipolar cells and which lies ventrally of the dorsal horn and dorsally and laterally of the canalis centralis. This region is both in Weigert and Nissl preparations very clearly distinguished structurally from the dorsal horns, having a more open reticular structure and larger rather numerous cells. This structure is characteristic of the corresponding region, *i. e.*, the area adjacent to the floor of the fourth ventricle, dorsally of the fasciculus longitudinalis dorsalis, far cephalad under the lobus vagi, and here it contains, besides the medium sized cells mentioned, the very large cells of the nucleus ambiguus. These latter cells extend nearly to the caudal end of the commissura infima (683), where they terminate abruptly.

For this nucleus of the spinal cord laterally of the canalis centralis I shall adopt the name "paracentral nucleus" from Onuf and Collins ('98). The nucleus ambiguus seems to be a specialized cranial portion of the paracentral nucleus.

At the level of the first spinal nerve this reticular area of the paracentral nucleus has spread out laterally and now occupies the region dorsally and laterally of the canalis centralis, the "intermediate zone" of Onuf and Col-

lins, which in higher animals contains the lateral cornu, Clarke's column and other structures now commonly associated with visceral nerves. The cells of the ventral cornu lie farther ventrally. At the level of the paracentral nucleus, but much farther laterally there runs through the spinal cord another ill-defined area of "ground substance" containing few medullated fibres and occasional very small cells (Fig. 16, *l*). This area, like the ventral cornu, is more or less obscurely connected by strands of non-medullated (mainly spongiplasmic) fibres with the other grey matter of the spinal cord. There is no connecting bridge of cells in the case of the lateral area.

In the striped mullet, Mugil cephalus L., which is closely related to Menidia and a young specimen of which (3 cm. long) was cut for comparison, we find the relations of the lobus vagi and its fibre connections very much as above described, with, however, the spinal portion more highly developed and more clearly differentiated. The caudal ends of the lobi vagi contract toward the ventricle and are continued into the spinal cord without an appreciable break. They finally lie closely appressed between the dorsal cornua and the ventricle and now numerous medullated fibres appear in them, a part or all of which cross dorsally of the canalis centralis after the fourth ventricle has closed, thus entering the commissura infima. A small bundle of medullated fibres is continued caudad of the commissure to about the level of the first spinal nerve. Its fibres gradually disappear, leaving the bundle in different directions, chiefly laterally and ventrally.

The motor nucleus of the IX and X (nucleus ambiguus) is very large and its cells are arranged in the characteristic dense rosettes. It extends far caudad beyond the end of the lobus vagi, maintaining the same position, *i. e.*, laterally and dorsally of the canalis centralis, until the level of the first spinal nerve. Here it ends abruptly, as in Menidia. In the mullet the ventral cornu cells extend cephalad considerably farther than the terminus of this

nucleus and the scattered cells which in Menidia lie laterally of the canalis centralis and in the neck of the ventral cornu (paracentral nucleus) also extend farther cephalad and form a much more compact nucleus. All of these structures may be seen in a single transection (Fig. 15), which passes through the caudal part of the nucleus ambiguus and the cephalic part of the ventral cornu, while the paracentral nucleus and the lateral recticular area extend a considerable distance farther cephalad. At the level of the first spinal nerve the paracentral nucleus has come to lie a little farther dorsally than in the figure so that, instead of lying ventro-laterally of the central canal, it lies laterally of it; *i. e.*, it occupies the position corresponding to that of the nucleus ambiguus of the medulla oblongata, and in this position I have followed it as a well-defined nucleus through nearly the entire length of the spinal cord. There is no break in the continuity of the cellular strand, though in parts of the trunk its cells are more numerous than in other parts. Its cells are throughout of the same large size as at first, while the cells of the dorsal cornu are very small. The latter are embedded in a dense gelatinous stroma, while the cells of the paracentral nucleus lie in a loose reticulum, in this again agreeing with Menidia.

I have described at length these structures of the spinal cord in this connection because they seem to me to be related to the communis system of the oblongata. In the fishes the communis system, as I have defined the term, is obviously concerned very largely with visceral sensations and is very intimately related to the viscero-motor apparatus.

Morphologically its terminal nuclei are not the most dorsal structures of the oblongata, as sometimes stated. Haller, for example ('96, p. 65), agrees with Goronowitsch in regarding the lobus vagi as the continuation of the dorsal cornu. It "occupies a position in the oblongata (dorso-

median part) which corresponds to that of the medial part of the dorso-lateral column of the cord."

In judging of the correctness of this assumption we must first determine what is the nature of the movement by which the canalis centralis has expanded to form the fourth ventricle. Remembering that the roof-plate of the nerve tube is membranous in the embryonic condition and is typically so in the adult, any massive structure appearing in it being a secondary ingrowth from the sides, it would appear that the membranous roof of the fourth ventricle represents this roof-plate and is represented in the spinal cord only by the floor of the dorsal fissure. The fourth ventricle is formed, then, by the dorsal and lateral expansion of the canalis centralis so that dorso-median structures of the cord become dorso-lateral structures of the oblongata. This is clearly shown by the course of the spinal V tract, which is unquestionably the cranial continuation of the dorsal horn (or at least of its general cutaneous portion). It is, then, obviously impossible to homologize structures lying in the floor of the fourth ventricle with those lying dorsally of the canalis spinalis, and this is what Goronowitsch and Haller attempt. The lobus vagi is developed in the floor of the fourth ventricle mesially and hence morphologically ventrally of the spinal V tract and the corresponding position in the cord must lie ventrally of the dorsal cornu. The spinal representative of the communis system of the head (visceral sensory), if such a component exists in the trunk, should have its terminal centre in the dorsal part of the intermediate zone, while the viscero-motor centre should occupy the ventral part of that zone.

This intermediate zone in higher animals contains the lateral cornu, Clarke's column and other structures now

commonly regarded as associated with the visceral nerves. Among these structures is the paracentral nucleus. This is regarded by Onuf and Collins ('98) as a motor splanchnic centre. In the cat it resembles very closely the nucleus to which I have given the same name in the fishes. I am willing to hazard the conjecture from the facts already in hand, that this intermediate zone of the fishes is in the broad view a visceral centre and homologous with the visceral centre of the region of the lateral cornu of the mammals. In the fishes the disproportionate size of these nuclei, as compared with the dorsal and ventral cornua, is not surprising, in view of the more important rôle which visceral nerves play in these animals. The paracentral nucleus of these fishes apparently corresponds to the "median nerve cells" of Kölliker ('96, p. 165). Some of the cells described by Van Gehuchten ('95, pp. 118 and 123) in this region of trout embryos probably belong to this nucleus.

Returning now to the oblongata, the homologies can be fixed here with more certainty. The pre- and post-vagal portions of the fasciculus communis in Menidia obviously represent the fasciculus communis of the Amphibia. It is certain that most of the pre-vagal fibres terminate in the lobus vagi. The diffuse terminal nucleus of the Amphibia (the spinal nucleus) has been compacted and enormously hypertrophied in the fishes, being represented in the lobus vagi (which, however, may contain other structures also) and in some fishes the "lobus trigemini" as well. The scattered cells about the lower part of the spinal portion of the fasciculus communis may represent a part of the original spinal nucleus which has not been absorbed into the lobus vagi.

The homologies with the mammals seem to be not less

clear. The fasciculus communis in the restricted (*i. e.*, the original) sense in its typical form as we find it, for example, in the Amphibia, conforms very closely to the fasciculus solitarius of mammals and birds, as has been pointed out by Strong. The same homology will hold in the fishes, with this difference, that all of the communis fibres of the vagus, and in some fishes of the glossopharyngeus as well, enter their terminal nucleus directly, without participating in the formation of the longitudinal tract known as the fasciculus communis. The relations of the terminal nuclei are rather more complicated. In the Amphibia, the spinal nucleus is the more important, the chief IX+X nucleus being relatively small. In the bony fishes the spinal nucleus has been either greatly reduced, or, more probably, fused with the chief nucleus (lobus vagi), which suffers more than a corresponding enlargement. In the mammals both nuclei are present and well developed. We know from Kölliker's work ('96, p. 246) that the fibres of the fasciculus solitarius of mammals give off collaterals into the substantia gelatinosa surrounding this tract and there probably come into relation with the cells of that region, which thus constitute a "spinal sensory nucleus of X," (Van Gehuchten, '97, p. 483). The sensory IX+X nucleus of mammals is represented in the lobus vagi of fishes, though it does not follow, of course, that the two structures are exactly equivalent.

This way of looking upon the sensory IX+X nucleus as merely a specialized portion of the spinal nucleus of the vagus or nucleus of the fasciculus solitarius receives the strongest support from the recent work of Cajal ('96, p. 44) by the Golgi method. In discussing the sensory terminal apparatus of the IX+X nerves of the new-born mouse, he writes: "There are, therefore, in this animal, not two

sensory terminal clusters, nor two separate portions for the two nerves. A single root, common to both nerves, passes over into the fasciculus solitarius without loss of any fibres, in such a way that between the upper, or chief nucleus, and the lower, or descending nucleus, there is no distinction aside from that of position."

The preceding considerations, it seems to me, remove the difficulties raised by Kingsbury, regarding the homology of the fasciculus solitarius of mammals with the fasciculus communis of the Ichthyopsida. He questions this homology ('95, p. 173) because, among other reasons, "by considering the *fasciculus communis* as representing the *fasciculus solitarius* alone, we leave unaccounted for the larger end-nidus of vagal sensory fibres. Strong, apparently, confounds this with the end-nidus of the *fasciculus solitarius*." This position he reiterates in his later paper ('97, p. 31). There is, it is evident, a certain amount of confusion in the recent literature regarding this homology. It is equally evident that this confusion is largely a matter of definition. If we use the terms fasciculus communis and fasciculus solitarius in the narrow sense, as longitudinal fibre tracts, and if we rigidly define the related structures, especially the terminal nuclei, and take into account the various transformations which the latter undergo in different classes of vertebrates (particularly Cajal's results cited above), it appears that in the broad view the fasciculus solitarius and its related structures in the mammals are, taken as a whole, homologous with the fasciculus communis and its related structures in the Ichthyopsida, though if we should attempt to draw up a detailed comparison, the various elements would doubtless not be exactly equivalent in the two groups of animals. Indeed, the fasciculus communis is, as we have

seen, by no means exactly equivalent in the different groups of the Ichthyopsida even.

In the fishes the post-vagal fasciculus communis is feebly developed and of obscure significance. It is chiefly, if not wholly, composed of secondary fibres from the lobus vagi and not of root fibres, so that it cannot be compared with the spinal V and spinal VIII tracts, nor wholly with the corresponding tract in the higher vertebrates (spinal portion of the f. solitarius). Yet its relations to the commissura infima Halleri and the nerve cells therein contained, suggest very strongly Cajal's recent description of the "commissural nucleus" of the mouse ('96, p. 46) which is as follows:

,, Die Fortsetzung der gemeinsamen Wurzel des Vagus und Glossopharyngeus bildet, wie gesagt, den Fasciculus solitarius. Dieses Bündel wird nach innen und hinteren von einer Columne grauer Substanz begleitet, in welche es nach Kölliker eine Unzahl fein verzweigter Collateralen aussendet, die, wenigstens bei wenige Tage alten Mäusen, niemals bis zur Substantia gelatinosa des Trigeminus vordringen. . . . . Dicht unter dem Ependym nähern sich die Solitärfascikel der Raphe und ihre grauen Terminalmassen vereinigen sich hier in einem Central- oder Mittelganglion, das wir *Commissurenkern* benennen wollen.

,, Drei Viertel der Fasern des Fasciculus solitarius enden, indem, sie sich kreuzen, in diesem Ganglion. Unter demselben besteht ausserdem ein kleines, bis über die Pyramidenkreuzung hinaus verlängertes Bündel, das anfangs in einer, vor dem Kern des Burdach'schen Stranges gelegenen grauen Masse, späterhin, nachdem letzterer verschwunden, im inneren Theil der Basis des Hinterstranges des Cervicalmarks anzutreffen ist. Wäh-

rend dieses intracervicalen Verlaufs sendet genanntes Bündel vereinzelte Collateralen aus, die sich nach innen zu in einen kleinen, ungenau begrenzten, sehr dicht an der hinteren Commisur gelegenen Herd grauer Substanz verzweigen."

A comparison of this description and the accompanying figures with the relations in the fishes which I have examined (including Mugil, Haploidonotus and Fundulus) very strongly suggests that the commissura infima Halleri contains the homologue of the commissure of Cajal's *Commissurenkern*. I am inclined to accept this homology, recognizing, however, that the commissura infima contains other fibres than those pertaining to the fasciculus communis. Whether the cells found in this region in fishes are homologous with the cells of the commissural nucleus of the mouse, I would not venture to guess.

Mayser ('81, p. 296) recognized the intimate relation of the commissura infima in the cyprinoids with the lobus vagi and the vagus nerve. The caudal portion of the commissure he inclines to regard as in part a decussation of the spinal V tract and in part a commissure of the dorsal cornua of the spinal cord. This I can confirm; *i. e.*, I find medullated fibres entering the commissure from the cephalic portion of the nucleus funiculi. They are probably secondary fibres. Niedzvietzky ('97, p. 542) has more recently described in the rabbit a commissure containing medullated fibres between the lower or caudal ends of the two fasciculi solitarii. A similar condition has been previously noted by Koch ('92) in the bird.

The homologies proposed in the preceding section may be summarized as follows:

(1). The fasciculus communis and its related structures (including the lobus vagi and the chief vagus roots)

of the fishes are as a whole homologous with the fasciculus solitarius and its related structures (including the sensory IX + X nucleus and the chief sensory vagus roots), excluding motor and general cutaneous elements in both cases.

(2). In the fishes the pre- and post-vagal fasciculus communis represent the fasciculus solitarius of the mammals, though the parallelism is probably not exact.

(3). The terminal nucleus of the fasciculus solitarius or the "spinal nucleus of the vagus" of the mammals has in the fishes been for the most part absorbed by the lobus vagi. Its caudal portion may persist, however, behind the commissura infima.

(4). The sensory IX + X nucleus of the mammals is merely a specialized portion of the nucleus of the fasciculus solitarius, and, accordingly, is represented with the latter in the lobus vagi of the fishes.

(5). The transverse fibres of the commissural nucleus of Cajal are probably homologous with the fibres of the spinal portion of the fasciculus communis, which appear in the commissura infima Halleri of the fishes.

Since this discussion was written and submitted for publication two papers have appeared which have an important bearing upon the homologies proposed. Both Van Gehuchten's researches upon the real origin of the cranial nerves (*Journal de Neurologie*, 1898) and Bunzl-Federn's paper on the central origin of the vagus (*Monats. f. Psychiatrie u. Neurologie*, V., 1, Jan., 1899) give the results of degeneration experiments by the Nissl method after resection of the vagus. Van Gehuchten shows that section of the vagus roots in the rabbit results in chromatolysis of cells throughout the dorsal or chief vagus nucleus. He concludes that this nucleus is wholly motor and confirms this by Golgi preparations. Marchi preparations showed that the sensory nucleus of the vagus is confined to the cells accompanying the fasciculus solitarius, the "spinal nucleus of the vagus." This would confirm in some degree the results of Forel ('91); but it is significant that Bunzl-Federn, also working upon the rabbit by the same method, and getting essentially the same anatomical results, does not admit that the dorsal nucleus is entirely motor.

If Van Gehuchten's results stand, it would require modification of the homologies proposed above to this extent: The chief, or dorsal, vagal nucleus of mammals would be a viscero-motor centre and the homologue of the sensory portion of the lobus vagi would have to be sought in the "spinal nucleus of the vagus" of mammals only.

Attention may also be called to the fact that the series of transections of the medulla of the rabbit given by Van Gehuchten (pp. 294–295) shows that the dorsal vagus nucleus and the fasciculus solitarius run down into the intermediate zone rather than into the dorsal horn region, thus agreeing with our findings in the fishes.

If we regard the fasciculus communis as the intracranial continuation of the visceral system of the "intermediate zone" of the spinal cord, as suggested above, we should expect to find in it or intimately associated with it many kinds of fibres, both afferent and efferent, for the various visceral functions. Such indeed is clearly the case, though we cannot as yet effect their analysis centrally. These being the primitive elements of the communis system, it follows that the fibres from the taste buds and terminal buds of the skin are phylogenetically later acquisitions, developed as a direct result of advancing cephalization. It is a significant fact that fibres from terminal buds are never found in the spinal nerves. Even in an extreme case, like the free tactile rays of the pectoral fins of the Triglidæ, which receive a surprisingly rich innervation from enormously hypertrophied spinal nerves and which by analogy with the barbels of other fishes we should expect to find covered with terminal buds, the most patient and skilful application of modern nerve methods (Morrill, '95) failed to reveal any terminal buds or other similar specialized organs in the areas supplied by these spinal nerves. On the other hand, whenever terminal buds occur on the bodies of fishes, as they often do over the entire surface, they are always innervated by recurrent nerves from the head. The terminal

buds of the trunk, too, we have reason to believe, have grown back into it from the head, as the organs of the lateral line are known to do. We have no evidence that the terminal bud system was primitively present in a metameric way in the trunk, but, as in the lateral line system, all the evidence at hand points to its cephalic origin. The theoretical problems connected with the communis system are further discussed in Section 12.

The secondary connections have not been fully worked out for any of the cranial nerves. In the case of the vagus, as with the acustico-lateral nerves, these connections are of two types, crossed and uncrossed. The uncrossed fibres, or secondary vagus bundle, in the sense of Mayser, gather mesally and ventrally of the spinal V tract and maintain this relation up to the exit of the latter from the brain, when they pass directly up into the cerebellum (Figs. 17, 18, 19, *Sec. X*). The crossed fibres, after reaching the opposite side through the commissura accessoria, enter the tractus bulbo-tectalis (*tr. b. t.*) and a large part, if not all of them, reach the optic tectum.

IV.—The Motor Components.

For the description of the motor nuclei the reader is referred to the accounts of the motor nerves in the following sections. Some points of a more general morphological interest regarding these components have been suggested in the preceding pages of this section. It need only be added here that I confirm, in general, the division of the motor cranial nerves in two series, somatic and visceral. They all belong to the latter type in the fishes save the eye-muscle nerves. The nuclei of the branchio-motor type, viz., nucleus ambiguus, motor VII and motor V, I consider to be cranial differentiations of a

strand of viscero-motor cells of the spinal cord running along the lateral and ventro-lateral side of the canalis centralis, the paracentral nucleus of Onuf and Collins.

Fürbringer ('97, pp. 664 and 680) assigns to these paracentral cells (his ventro-lateral series) and their fibres which emerge with the dorsal roots (his lateral fibres) the viscero-motor function throughout the Vertebrata, including the Cyclostomata and Amphioxus. The visceral component is larger in cyclostomes than in higher vertebrates. These cells he also directly compares with the viscero-motor nuclei of the oblongata.

V.—Summary of Section 3.

The general cutaneous nerves enter the brain by the V and X roots. The trigeminal fibres terminate in part in a chief trigeminal nucleus mesally of the point of entrance of the root, while the larger part pass caudad as the spinal V tract to terminate, after receiving the general cutaneous component of the vagus, in the n. funiculi of the spinal cord. This system is, therefore, the direct cranial representative of the dorsal cornu of the cord.

All nerves of the acustico-lateral system (r. lateralis vagi, VIII, dorsal and ventral lateralis roots of VII) terminate in the tuberculum acusticum, which is intimately related to the cerebellum. This system has also ascending (cerebellar) and descending (spinal VIII) roots.

The communis system is represented in the VII, IX and X nerves. The terminal nucleus for all of its fibres lies in the lobus vagi. This system is very completely isolated and unified in the head, and yet is apparently related to a sensory system of the trunk, closely associated with the spinal viscero-motor centres (intermediate zone). The communis system was primarily a viscero-sensory

(entodermal) mechanism. This is its sole function in the trunk. In its cranial portion it also supplies taste buds and terminal buds (ectodermal organs). The communis system as a whole corresponds with the fasciculus solitarius of mammals, together with its associated structures (sensory vagus nucleus, etc.), though the comparison is not exact. Each of these tracts is related to a dorsal commissure, the commissura infima Halleri in the one case and the fibres crossing in connection with the commissural nucleus of Cajal in the other.

Section 4.—The Spinal Nerves.

I.—The Fourth Spinal Nerve.

Our examination begins with the fourth spinal, which may be regarded as the first typical trunk nerve. The dorsal and ventral roots, of which the latter is the larger, emerge by distinct but closely approximated foramina through the base of the neural arch of the third free vertebra. The roots, foramina and ganglion all lie in the same transverse plane. The dorsal root (*4 sp. d.*) is composed wholly of fine fibres, the ventral root chiefly or wholly of coarse ones. From the ganglion are given off two minute dorsal rami, a medium-sized medial ramus and a large ventral ramus, besides the r. communicans with the sympathetic trunk.

The *ventral ramus* (*r. v. 4*) pursues the typical course. The *medial ramus* (*r. m. 4*), like the ventral, contains both sensory and motor elements. It passes laterad and caudad, not in the intermuscular septum between the dorsal and the lateral musculature, but through the lateral part of the dorsal musculature, following for part of its course one of the intermuscular bones, which it finally

crosses. Just before reaching the skin it breaks up around the r. lateralis vagi to supply the muscles and skin of that immediate neighborhood.

Baudelot and many others regard the ramus medius as the homologue in the spinal nerves of the r. lateralis vagi. This is undoubtedly an untenable hypothesis; and the r. lateralis cannot be regarded as a collector for the spinal nerves in the sense so commonly assumed. The significance of this anastomosis, where it exists, is discussed at the close of Section 12, I.

The two *dorsal rami* are quite distinct in origin and nature. One (*r. com. 4*), of fine fibres, arises from the ganglion and, directly dorsad on the outer surface of the neural arch of the vertebra, joins the ramus spinosus of the third spinal nerve (*r. sp. 3*), the compound nerve then running dorsally in the intermuscular septum between the general dorsal musculature and the interspinal muscles (the mm. supra-carinales of Owen), where it joins the r. lateralis accessorius. This nerve is the *r. communicans* of Stannius and is apparently exclusively sensory. The other dorsal ramus (*r. sp. 4*) arises from the motor root and is apparently exclusively motor. It effects similar relations with the r. communicans of fifth spinal nerve and then, as before, the mixed nerve thus formed joins the r. lateralis accessorius, previously, however, sending numerous fine branches into the dorsal musculature. This is the *r. spinosus* of Stannius.

This, it appears from the literature, is the typical arrangement of dorsal rami in fishes. (Compare especially, Owen, '66, Vol. I, p. 308.) The fact that the cephalic one of these rami is sensory and the caudal one motor would seem to be correlated with the rhythmical movements of the body in swimming. Thus the sensory

stimuli arising in the contracting segment and transmitted to the spinal cord through the r. communicans might be of use in regulating the motor impulses to be transmitted to the next following segment through the r. spinosus. The anastomosis with the r. lateralis accessorius is in every segment examined a very broad one, the entire sensory component of the dorsal spinal ramus passing bodily into the r. accessorius. See the latter nerve for further mention.

The relations of visceral fibres to the spinal nerves have not been investigated, as my methods are not adapted for that research.

II.—The Third Spinal Nerve.

The origin of the third spinal nerve resembles that of the fourth except that the roots are larger; they emerge in the same way by separate foramina in the second free vertebra, and exhibit the same difference in the calibre of the fibres. The coarse ventral fibres, as before, can be traced through the ganglion. Of the dorsal rami, the *r. spinosus*, *r. sp. 3* (wholly motor), and the *r. communicans*, *r. com. 3* (wholly sensory), are as in the last case; so also the *r. medius*, *r. m. 3*, save that the number of its sensory fibres is smaller.

The *ventral ramus* (*r. v. 3*) is large, the sensory portion being more than three times the size of the motor, which is of about the average size for a spinal nerve. It gives off immediately a minute twig for the dorsal musculature (not shown on the plot), and then runs under the dorsal musculature to the lateral edge of the latter, where it breaks up into three branches. Two of these, which are composed of fine fibres, run caudad and laterad, the first running up over the depressor of the pectoral fin and

between that muscle and the dorsal musculature, the second behind the insertion of the depressor, and both distributing to the surface of the fin. Their ramuli run out parallel with the fin rays, those of the first branch supplying the skin of the middle portion of the fin, those of the second branch the skin of the ventral portion.

The third branch is the true ventral ramus, containing all of the motor fibres and the remainder of the sensory fibres. Having separated ventrally from the other branches, it receives a small coarse-fibred twig from the brachial plexus, then continues ventrad to supply the ventral musculature (*v. m.*) and the skin lying immediately behind the pectoral girdle. The three branches of the ventral ramus are crossed externally near the point of their separation by the other fibres for the pectoral fin derived from the brachial plexus; but the third spinal has no connection with the brachial plexus save the small motor twig received by the ventral ramus, though on the opposite side of the specimen plotted the relations are somewhat different; see the account of the second spinal.

III.—The Second Spinal Nerve.

The roots of the second spinal nerve are larger than those of any of the succeeding nerves, the dorsal root (*d. 2*) being somewhat larger than the ventral. The two roots pass out through a common foramen in the neural arch of the first free vertebra. The two *dorsal rami* are like those of the third spinal nerve; the *ramus medius* (*r. m. 2*) also has the same relations except that it appears to contain only motor fibres.

The large *ventral ramus* (*r. v. 2*) under the lateral edge of the dorsal musculature is joined by a large bundle of coarse and fine fibres from the mixed ventral ramus of the

first spinal (*r. v. b+c*) and at the point of union there arises from the second spinal the small bundle of motor fibres which has been mentioned as going down to join the ventral ramus of the third spinal. The mixed trunk now gives off about six small branches of coarse fibres for the depressor of the pectoral fin, two of which are drawn upon Fig. 3 (*dep.*) These motor fibres are derived chiefly from the first spinal, though a few seem to come from the second. Those from the first spinal arise certainly mainly and probably exclusively from its caudal motor root, *c*.

The mixed trunk now turns laterally, through the depressor muscle and through a foramen in the scapula just behind the caudal edge of the cleithrum, the fibres meanwhile arranging themselves in three groups, the sensory fibres from the first spinal lying dorsally, the sensory fibres from the second spinal ventrally and the motor fibres between. The latter are derived chiefly from the first spinal, though a small bundle from the second is included. The sensory fibres from the first spinal (*cut 1*) remain distinct from those from the second spinal and pursue a very tortuous course to the skin of the side of the body behind the operculum and overlying the levator muscle of the pectoral fin. The cleithrum here consists of two broad lamellæ with the origin of the levator between them. This nerve runs first cephalad along the outer face of the inner lamella, then laterally through the muscle and then caudad along the inner face of the outer lamella of the cleithrum to the skin behind the latter.

A twig containing motor fibres from the first spinal leaves the trunk at the same point as the last and supplies the dorsal portion of the levator muscle (*lev.*)

The sensory fibres from the second spinal turn dorsad, the remaining motor fibres from the first and second

spinals turn ventrad, along the outer face of the inner lamella of the cleithrum. In the latter nerve the fibres from the two sources cannot be separately followed, but all enter the ventral portion of the levator of the pectoral fin (*lev.*) The sensory fibres last mentioned (*f. d. 2*) pass to the pectoral fin and break up into several ramuli which run out parallel with the fin rays, thus supplying the skin of the dorsal part of the fin, *i. e.*, the portion not innervated by the third spinal.

On the right side of the specimen upon which this description is based the composition of the brachial plexus is in one respect different. The middle portion of the pectoral fin is supplied by a nerve which has the same course over the depressor of the fin as the corresponding nerve on the left side, but which has a double origin. The greater portion of the fibres arise from the mixed ventral rami of the first and second spinals before that nerve has pierced the scapula; they can be traced back to the dorsal root of the second spinal nerve. Shortly after their separation these fibres are joined by a much smaller twig from the third spinal, which arises at a point corresponding to the origin of the main nerve on the opposite side and then, instead of going caudad to the fin, turns cephalad for a considerable distance to effect the anastomosis. Which of these two cases is to be regarded as typical for this species, it is impossible to state, as comparative data are lacking.

IV.—The First Spinal Nerve.

The first spinal nerve (Fig. 7) differs widely from the others and shows very plainly that it is formed by the incomplete fusion of at least two segmental nerves. There are four roots, two dorsal and two ventral, all well developed and the two pairs widely separated. It is obvious

from the sections that, in the adult, at least one vertebra has been fused with the skull, and through the portion of the cranium thus formed the roots of the first spinal emerge, the caudal pair (dorsal and ventral) through one foramen and the cephalic pair through another.

In conformity with the nomenclature of Fürbringer ('97), the cephalic pair of roots and the nerves issuing therefrom will be designated by the letter *b*, the caudal pair by *c*. The ganglia of the nerves *b* and *c* fuse to a certain extent, so that it is impossible to determine with precision from which source some the sensory fibres of the peripheral rami come. The motor roots of *b* and *c* do not, however, mingle, so that, though the relations of the numerous rami at their origins are very complicated, yet it is possible to trace each with precision to its root.

The ventral roots are much larger than the dorsal, and both roots of *b* are larger than the corresponding roots of *c*, the dorsal root *c* being very minute. As in the case of all of the spinal nerves, the dorsal root arises at the extreme dorsal side of the spinal cord, the ventral leaves at its extreme ventral side, the two roots emerging through the foramen at the same transverse level nearer the ventral than the dorsal side. Some of the fibres of the motor root *c* are clearly seen to arise from the fasciculus longitudinalis dorsalis (Fig. 16). Their ultimate source is unknown. This root has two kinds of fibres, about half of the number being less densely myelinated so that in Weigert preparations they stain a much lighter blue than the others. They are as large as, or even larger than the others which are of the usual character of motor fibres. Their significance is unknown, as they could not be traced far in either direction.

These roots and their rami are indicated in detail in

Fig 7, which is drawn from the same specimen plotted on Fig. 3, but to a larger scale. In this figure the sensory fibres are indicated in yellow, the ganglion being a lighter shade of the same; the motor fibres of *b* are drawn in light blue, the motor fibres of *c* in a darker blue.

Now taking up the rami arising from this complex in order, we have first a *ramus communicans* arising from the cephalic tip of the ganglion *b* (*r. com. b.*) consisting wholly of sensory fibres, and pursuing a typical course cephalad and dorsad over the upper surface of the supra-occipital bone. Leaving this bone, it then runs up in the intermuscular septum laterally of the interspinous muscles to join the r. lateralis accessorius in two strands (Fig. 3), the latter nerve having at this level turned mesally from its former lateral position to run in the same intermuscular septum, which position it maintains from this point caudad.

Close behind the last there arises a minute motor twig (*is. m. b.*), which pursues a course similar to that of a r. spinosus, though the true r. spinosus of this segment lies a little farther caudad. It supplies the interspinal muscles and does not join the r. lateralis accessorius, though it distributes near the latter nerve. The *true r. spinosus of b* (*r. sp. b.*), as it runs back over the supra-occipital bone sends a few fibres into the dorsal musculature and then joins the r. lateralis accessorius in the intermuscular septum in the typical manner, save that it does not anastomose with the r. communicans of the next following segment. It is exclusively motor.

The *r. medius b* (*r. m. b.*) arises between the two nerves last mentioned and contains motor fibres and a smaller number of sensory. Its stem at once divides into two branches, each of which takes some of both motor and sensory fibres and penetrates the dorsal musculature,

within which one runs cephalad and laterad, the other caudad and laterad. The former, having reached almost to the lateral edge of the dorsal musculature, sends a small motor twig farther forward, then turns abruptly caudad until, still within the same muscle, it joins the caudal branch. Here the sensory fibres of both branches unite and, separating from the motor fibres, run up in several bundles to supply the skin about the lateral line near its junction with the supra-occipital commissure. In their course toward the skin these sensory bundles run in an intermuscular septum which is occupied by that limb of the extra-scapular bone which articulates with the cranium, and follow, some the outer, some the inner face of that bone. Both branches of the r. medius *b* give off motor fibres for the dorsal muscle throughout their entire courses, and after their union and the separation of the sensory fibres, their fibres at once distribute to the lateral portion of that musculature.

The large *ventral ramus of b* (*r. v. b.*) takes the remaining motor fibres from that root and also a considerable bundle of sensory fibres. That the latter come from the dorsal root *b* is from every standpoint very probable, nay, almost certain, yet the sections do not afford an absolute demonstration, as the ganglia of the two roots cannot be sharply separated. The mixed ramus runs out under the dorsal musculature, and is there joined by the ventral ramus *c* (*r. v. c.*), with which its further course will be described.

There is no *r. communicans c.* Some fibres from the dorsal root *c* may, however, go out with the r. communicans *b*, the double nature of the anastomosis of that nerve with the r. lateralis accessorius offering a suggestion of such a condition.

The *r. spinosus c* (*r. sp. c.*) is perfectly typical. It runs back over the cranium and first free vertebra and effects the anastomosis with the r. communicans of the second spinal and the r. lateralis accessorius in the septum laterally of the interspinal muscles in the usual manner. It is composed of motor fibres, and just as it leaves the ganglion it gives off a motor branch dorsally for the dorsal musculature.

The *r. medius c* (*r. m. c.*) arises just cephalad of the r. spinosus. It is a small nerve and, like the corresponding nerve of *b*, it contains chiefly motor, with a few sensory fibres. It runs for a considerable distance cephalad in the ganglion, which it leaves close behind the r. medius *b*. It follows the latter nerve, lying ventrally of it, for some distance, though there is no anastomosis between them. It, however, goes much farther caudad than that nerve, running for a time parallel to the first intermuscular bone, and at the level of the third branch of the r. lateralis vagi (765) it crosses the second twig of that nerve (*r. l. 2*) and anastomoses with it. Some of its sensory fibres apparently go out to the skin with that twig, others independently a little farther caudad, the motor fibres having already been given off. Thus it appears that the r. medius of both *b* and *c* conforms to the typical arrangement of the spinal nerves in that the sensory fibres are distributed to the skin about the lateral line.

The ventral ramus of c takes the remaining fibres from the ventral root *c* and a smaller number of sensory fibres. It is smaller than that of *b*. It follows the latter nerve out under the dorsal musculature and soon joins it, the mixed trunk soon thereafter being joined by the r. ventralis of the second spinal nerve. At the point of union with the latter nerve a small motor twig (derived, it is

clear, from the ventral ramus *c*) separates dorsally to enter the depressor of the fin (Fig. 7, *dep.*) and just before this point the so-called hypoglossus nerve (the *r. cervicalis*, Fürbringer, *r. cerv.*) separates from the first spinal. This nerve draws off all of the sensory fibres of the r. ventralis *b* and a smaller number of motor fibres from the same source. It turns directly ventrad from its point of origin, running down in the walls of the abdominal cavity in the manner typical for ventral spinal rami. It descends first along the caudal face of the m. pharyngo-branchialis externus, then lower down in the same relation to the m. pharyngo-branchialis internus. Midway of this course two or three minute twigs of fine fibres are given off which run in a lateral direction along the caudal face of the m. pharyngo-branchialis externus to the skin which covers the cleithrum laterally. Having reached the cleithrum, the remaining fibres of the r. cervicalis turn cephalad along the inner surface of the m. pharyngo-branchialis at its origin from that bone. The origin of the pre-zonal ventral musculature from the cleithrum interdigitates with that of the pharyngo-branchialis, and the r. cervicalis, following the inner border of the latter muscle, comes to lie embedded between two slips of ventral musculature (m. sterno-hyoideus). Here a fine fibred cutaneous twig is given off ventrally to supply the skin of the ventral surface under the united tips of the two cleithra; the coarser motor fibres (Fig. 3, *m. shy.*) ramify through the substance of the sterno-hyoideus, which they innervate. No fibres from this nerve enter either of the pharyngo-branchial muscles, nor is there any anastomosis with the vagus, though the former condition is said to prevail in Amiurus (Wright, '84) and in the lower fishes (Fürbringer, '97) and the latter is figured by

Fürbringer ('97, Plate VIII, Figs. 5 and 6) in Esox and Mullus.

In another specimen from the one just described and figured the composition of the r. cervicalis is somewhat different. In addition to sensory and motor fibres from the first spinal nerve *b*, it receives also a large number of sensory fibres from the ramus ventralis *c*. The latter nerve has a larger proportion of fine fibres than in the specimen figured and it is not improbable that the fibres which it gives to the r. cervicalis are derived from the dorsal root *b*. In this specimen, as in the other, the motor fibres of the r. cervicalis clearly all come from the motor root *b*.

V.—Comparative Review of the First Spinal Nerve.

The cervical nerve corresponds to the cervical, *i. e.*, prezonal, plexus of the Selachii and in part to the n. hypoglossus of the higher vertebrates. Of course in the latter case the sensory element here present will have to be excluded. The results of Harrison ('95) go to show that the sterno-hyoid muscle of the salmon has the same origin ontogenetically as the tongue-muscles of the higher forms, that is, it is a true somatic muscle, derived from the lateral muscle plates. The nerve supply is therefore unquestionably homologous. Nevertheless the r. cervicalis of the bony fish cannot be homologized without reserve with either the cervical plexus of the Selachii or the hypoglossus of higher forms, for in both of these cases the nerves in question supply not only the post-hyal, but also the pre-hyal ventral musculature, while in the bony fish the latter is, as we shall see, absent. These points are again referred to in our account of the m. genio-hyoideus, Section 7, IV, 5, *iv*. For further critical and historical

discussions the reader is referred to Kupffer's excellent digest ('96) and to Fürbringer's great monograph ('97).

It should be noted that neither of these works clears up the problems connected with the pre-hyal ventral musculature of teleostomes and that in regard to another important moot question they are absolutely contradictory, viz., the morphology of the hypoglossus region of the cyclostomes. In the case of Petromyzon Kupffer describes the ventral musculature of the head as innervated from the vagus and not from the "hypoglossus" or first spinals, as in most other vertebrates. He thought that the ventral musculature of Petromyzon is of dermal origin and that it is not derived from the lateral muscle plates, as is the case with the "hypoglossus musculature" of other vertebrates. This would explain the innervation from the vagus instead of the first spinals and there would be no true hypoglossus in Petromyzon, for the corresponding musculature is wanting.

Subsequenty Neal ('97), working under Kupffer's direction, has re-opened the question, and he finds that the ventral musculature of the head is developed in Petromyzon in exactly the same way as in other vertebrates and is homologous throughout the series. He therefore concludes that the r. recurrens vagi of Petromyzon is homologous with the hypoglossus of higher vertebrates, while the so called hypoglossus of the older writers on Petromyzon is composed of true spinal nerves.

Fürbringer, however, comes to a quite different conclusion. He finds ('97, p. 597), both in Ammocœtes and in adult Petromyzon, that the r. recurrens vagi is improperly named, for it contains no vagus fibres whatever; it is rather a r. recurrens spinalis, only secondarily bound up with vagus fibres and clearly separable from them. Petromyzon, therefore, conforms to all of the other vertebrates, of which Fürbringer has studied types of every class, in that the hypoglossus musculature is innervated by the spinals and the spinals only, the vagus never participating. Alcock ('98, p. 150) fully confirms

Fürbringer's conclusions from a study of serial sections of Ammocœtes.

This is a matter of no small importance, for the innervation of the ventral musculature, including the pharyngo-clavicular muscles, (cleido-branchialis 5) from the spinal nerves in all vertebrates is the key-stone of Fürbringer's argument from comparative anatomy for the distinction of paleocranial and neocranial nerves, a distinction of the most fundamental importance for his scheme of the metamerism of the head. According to that scheme, the primordial cranial nerves, back to and including the IX+X+XI complex, in all forms above the cyclostomes belong to those segments which are comprised in the paleocranium of Gegenbaur and from this point upward are to be sharply separated from the spinal nerves, though secondary anastomoses of various forms may occur. Passing up the taxonomic series a progressively larger number of spinal segments become fused with the head and either wholly or partially degenerate. In no case do their nerves fuse intra-cranially with the paleocranial nerves; on the contrary, they simply atrophy and to their more or less modified vestiges the name "spino-occipital nerves" is given. This group of nerves is further subdivided into "occipital nerves," which have lost their spinal character and have become incorporated into the head so that they emerge through foramina in the cranium, and "occipito-spinal nerves," which, though they emerge behind the cranium, yet have suffered some modification, usually the reduction of the dorsal root. The general rule may be laid down, that among the adults of almost every class of vertebrates, the more primitive forms are characterized by more, the higher forms by fewer, of the spino-occipital nerves. The embryology in most cases where it is known recapitulates more or less completely the steps in this reduction.

In Myxine there are five or six, in Petromyzon two or three spinal nerves which lie cephalad of the first spinal of the lowest selachians (Notidanidæ). With the latter forms the paleocranium is completed and the formation of

the neocranium begins. The higher mammals have lost from five to six of the first spinal metameres as compared with the Notidanidæ, the hypoglossus nerve corresponding probably to the ventral root of the twelfth spinal segment of Myxine.

In the bony fishes there are, as a rule, no occipital nerves, the first occipito-spinal nerve (*a*) of the ganoids, etc., is lost, and the second and third occipital nerves (*b* and *c*) are present, usually more or less intimately united with each other and often with the next following, or first true spinal nerve (4 of Fürbringer's nomenclature, my second spinal). In most of the teleosts examined by Fürbringer the ramus cervicalis is formed chiefly from *b*, with the addition of a variable number of fibres from *c*. In one case it is formed, however, wholly from *b*, as in the specimen of Menidia figured.

It is interesting to note that in Menidia the process of cephalization has gone farther in this respect than in any teleost mentioned by Fürbringer; for the "occipito-spinal" nerves *b* and *c* of those teleosts have here become "occipital" nerves. That is, the corresponding vertebra has been fused with the skull and they therefore emerge through the cranium. This is a phenomenon difficult of explanation in view of the low taxonomic position given to Menidia among the teleosts. It may be a cenogenetic acquisition correlated with the excessive development of the ears and the cranial parts with which they are related. In any case it does not stengthen one's confidence in the spino-occipital nerves as guides to phylogeny.

This latter conclusion I find is confirmed by Allis' latest paper ('98). From dissections of Scomber made under his direction by Dr. J. Dewitz, he finds a condition of the spino-occipital nerves which conforms neither to Fürbringer's account of the teleosts nor to my findings in Menidia, but much more closely to Amia. The spino-occipital nerves *a*, *b* and *c* of Fürbringer are all present, though their roots, as in Menidia, emerge through the occipitale laterale of the skull and fuse into a single ganglionic complex. From this and other cases cited by Allis

it is probable that the teleosts typically possess an occipito-spinal nerve *a* in a reduced condition. Whether that nerve in Menidia has been lost or has fused with *b* I cannot state. I have not made sufficiently extensive comparative studies to speak with authority on these homologies, nor have my methods been adapted for this problem.

In connection with the fact that the spino-occipital nerve *a*, which is lost or reduced in the teleosts, is present both in lower and in higher forms—as a spinal nerve in most Selachii and Amphibia, and as an occipito-spinal nerve in some sharks and ganoids, in Holocephali, Dipnoi and many Amniota (Fürbringer, '97)—it is exceedingly suggestive to notice that it is the teleosts alone of all these forms which lack the true pre-hyal ventral musculature. The so-called genio-glossus of teleosts is supplied by the trigeminus and is quite certainly not derived from the hypoglossus, or ventral spinal musculature (see the discussion in Section 7, IV, 5, *iv*), and with the loss of this musculature the corresponding spino-occipital nerve has naturally also suffered reduction.

Haller ('96, p. 53) says that in Salmo the spino-occipital roots (his post-vagal nerve) do not participate in the innervation of the hypoglossus musculature, but distribute exclusively to the pectoral fin. The ventral musculature is innervated, according to this authority, by a branch from the vagus. It will probably prove that this description is inexact, and that either the vagus branch in question supplies the pharyngo-branchial muscles instead of the sterno-hyoideus or else that the vagus stem is joined by fibres from a spino-occipital nerve which was overlooked by Haller. His generalization (p. 56) that the hypoglossus nerve was primitively included with the vagus and has only secondarily been dissociated from it will certainly not stand in the light of our present knowledge of both higher and lower forms.

VI.—Summary of the Spinal Nerves.

The typical spinal nerve gives off a ventral ramus, a medial ramus and two dorsal rami. The ventral ramus contains motor and sensory fibres for the ventral musculature and skin. The ramus medius contains motor fibres for the dorsal musculature and cutaneous fibres for the skin in the vicinity of the lateral line canal. This nerve is in no sense comparable with the lateral line branches of the cranial nerves, and the term ramus lateralis would better be avoided in the case of the spinal nerves, as suggesting bad morphology. Of the two dorsal rami, the first (r. communicans) is sensory, the second (r. spinosus) is motor. Each r. spinosus anastomoses with the next following r. communicans and supplies the dorsal musculature near the median line, especially the interspinal muscles. The r. communicans joins the r. lateralis accessorius and doubtless innervates the overlying skin of the back.

The brachial plexus receives elements from both portions *b* and *c* of the first spinal nerve and from the second and third spinals. The pre-zonal portion of this plexus—the ramus cervicalis of Fürbringer—receives some of the motor fibres of the r. ventralis *b* and all of the sensory fibres of that ramus. The post-zonal plexus has a single ventral ramus (in the strict sense, *i. e.*, for ventral musculature and skin) which receives some of the sensory fibres from the ventral ramus of the third spinal nerve, all of the motor fibres of that ramus and a small number of motor fibres from the ventral ramus of the second spinal. The sensory ramus for the skin of the side of the body between the pectoral fin and the opercular cleft, contains all of the sensory fibres of the ventral ramus *c* of the first spinal and no others. The depressor muscle of the pectoral fin is innervated chiefly from the ramus ventralis *c*

of the first spinal, though it apparently also receives a few fibres from the r. ventralis of the second spinal. It consumes probably all of the motor fibres of the r. ventralis *c*. The levator muscle of the pectoral fin is supplied chiefly by the r. ventralis *b* of the first spinal, but partly by the r. ventralis of the second spinal. The skin of the pectoral fin is supplied by three branches; the dorsal part by a nerve which comes from the ventral ramus of the second spinal and includes all of the sensory fibres of that ramus; the middle part by a nerve which in one case comes from the ventral ramus of the third spinal, in another case from the ventral rami of both the second and the third spinals; the ventral part by a nerve which comes from the ventral ramus of the third spinal.

The composition of the brachial plexus may be tabulated as follows:

First spinal nerve.
- Ramus ventralis, *b*,
  - Sensory: supplies all of the sensory fibres of the r. cervicalis.
  - Motor: supplies all of the motor fibres of the r. cervicalis (for the m. sterno-hyoideus) and most of those for the levator of the pectoral fin.
- Ramus ventralis, *c*,
  - Sensory: supplies all of the fibres for the nerve for the skin between the pectoral fin and the opercular cleft.
  - Motor: supplies almost all of the fibres for the depressor of the pectoral fin.

Second spinal nerve, r. ventralis,
- Sensory: supplies dorsal part of pectoral fin and sometimes the middle part.

Motor: supplies some of the motor fibres of the first post-zonal ventral ramus (*sensu stricto*), a few fibres probably for the depressor of the pectoral fin and a small number for the levator of the pectoral fin.

Third spinal nerve, r. ventralis,

Sensory: supplies all of the sensory fibres of the first post-zonal ventral ramus, and sensory fibres for the middle and ventral portions of the pectoral fin.

Motor: supplies most of the motor fibres of the first post-zonal ventral ramus.

The first spinal nerve is a fusion of at least two complete segmental nerves whose motor components can be clearly distinguished from each other. All four of these roots emerge through the cranium, instead of behind it, as in other teleosts. The reduction in the teleosts of the first occipito-spinal nerve *a* is correlated with the absence in the teleosts of the pre-hyal ventral spinal musculature. Both this nerve and the corresponding musculature are found in vertebrates both above and below the teleosts in the taxonomic series.

Section 5.—The Nerves of the Vagus Group.

This section includes the account of the IX, X and XI nerves, including the r. lateralis vagi. The glossopharyngeus and the r. lateralis are anatomically quite distinct from the vagus; nevertheless they can best be described together. These nerves in Menidia conform in general to the usual teleostean arrangement, giving evidence in some features of specialization parallel with an elaborate branchial apparatus as a whole.

The vagus group, as here defined, contains general cutaneous, acustico-lateralis, communis and viscero-motor components. The general cutaneous component is very small, the acustico-lateralis and viscero-motor are of considerable size, but the communis fibres make up by far the larger portion of the complex. This latter system is, however, far less highly developed here than in some other teleosts, such as the cyprinoids, in which the vagal lobes attain so enormous proportions. The IX nerve receives communis and viscero-motor fibres; the X nerve proper receives communis, viscero-motor and general cutaneous fibres. The n. lineæ lateralis, conventionally associated with the vagus, has no obvious morphological relation to the other components of this complex.

I.—The Roots and Ganglia of the Vagus Group.

The roots of the IX and X nerves are quite widely separated, the former arising under the origin of the r. lateralis vagi. The sensory and motor roots of the IX nerve are distinct at their origins, though close together, and they unite immediately after leaving the brain. The various elements of the vagus (aside from the r. lateralis) are so intimately united that analysis by gross methods would be quite impossible.

1.—The Communis Root of the N. Glossopharyngeus.

The sensory root of the IX nerve emerges from the brain just dorsally of the motor IX and these two roots diverge from each other very rapidly in passing toward their respective centres. The communis root passes dorsally of the spinal V tract, the motor root ventrally of it. This sensory root runs directly inward as a close round bundle of very fine fibres until it reaches the fasciculus

communis, which runs along the lateral wall of the fourth ventricle. Its fibres turn caudad and enter this fasciculus, which in turn immediately enters the lobus vagi, as already described.

These fibres which enter the lobus vagi by way of the fasciculus communis do not appear to differ in any way from those which enter it directly from the vagus, for their central and peripheral relations are essentially the same. Indeed in those fishes which have larger lobi vagi the IX nerve enters the lobus directly without participating in the formation of the fasciculus communis, as in the cyprinoids (Mayser, '81) and in the gadoids and Raniceps (Stannius, '49).

The IX nerve emerges close under the origin of the r. lateralis vagi, its origin being covered by the ramus ampullæ posterioris of the VIII nerve. These latter fibres pursue an intracranial course as a thin flat band, running up until they lie in contact with the r. lateralis vagi. The IX root runs along the inner side of this acoustic root and finally crosses it ventrally, the two roots lying as close to each other as possible. There is certainly no extensive anastomosis between these roots and probably there is no exchange of fibres whatever, though from the nature of the conditions it is impossible to be sure of the latter point.

It can, however, be clearly made out that a very small bundle of communis fibres separates from the dorsal surface of the IX root before this nerve has crossed the VIII root. This bundle goes dorsad between the fibres of the VIII nerve, already mentioned, and the medulla oblongata and enters at once the lateralis root of the vagus. Its further course will be described with that nerve.

The IX root now continues to run directly outward, penetrates the ear capsule and then turns cephalad along the outer surface of the latter, lying ventrally of the vagus ganglion, but not at any time coming into contact with it. Here it is joined by the sympathetic chain, which accompanies it as far as the IX ganglion and then continues cephalad in the same direction.

The IX ganglion lies at the point where the nerve turns laterally and ventrally into its gill. A small sympathetic ganglion is applied to its proximal portion. The connection with the sympathetic chain and the anastomosing branch to the root of the r. lateralis vagi are the only connections which the glossopharyngeus has with any other nerves.

2.—The Communis Root of the Vagus.

The vagus proper arises by one great root (640–660), which contains three components intimately fused. The great majority of these fibres are communis fibres and pass directly into the lobus vagi, forming the outer fibrous layer of that structure. In this intracranial portion of their course these root fibres separate into two ill-defined tracts, a larger caudal root and a smaller cephalic as shown on Fig. 4.

Immediately upon entering the oblongata they cross the spinal V tract. This tract, which caudad of this level forms a cord-like protuberance on the dorso-lateral aspect of the oblongata, here breaks up into several large strands and sinks abruptly down to a more ventral position. The root fibres of the vagus interdigitate with these strands so that the relations here are rather confusing. In Fig. 4 all of the vagus fibres are conventionally drawn as if they crossed the inner and ventral face of the spinal V tract;

in fact the more cephalic of the root fibres run over the outer and dorsal surface of the spinal V tract.

The vagus ganglion seems single macroscopically, like the root, but microscopically it is clearly separable into four ganglia, as shown in Fig. 4, corresponding to the four branchial clefts innervated by this nerve. In each case the ganglion cells are separated from those of the adjacent divisions by the entering and emerging fibres, so that in Weigert preparations especially the discreteness of the ganglia is very conspicuous. This is slightly exaggerated by shrinkage during hardening. Nearly all teleosts, according to Stannius, exhibit such a branchiomeric condition of the vagus ganglia, though there is the widest variation in its amount. We have numerous steps to the condition in Raja (Shore, '89), where the widely separate ganglia lie each in its own branchial ramus. And this is especially interesting in view of Cole's more recent discovery ('96) that in Chimæra the IX nerve and the three branchials and the r. intestinalis vagi have quite independent origins and ganglia, like the IX in teleosts. The distinctness of the vagal ganglia in the present case may therefore be looked upon as vestigeal, and not merely as an adaptation to the existing branchial apparatus.

The fourth ganglion in Menidia (*g. X. 4+5*) is much the largest. It includes, besides the ganglion for the nerves of the fifth gill cleft, which are much smaller than the others of the series, the ganglion for the great visceral and œsophageal rami of the vagus. The ganglia for these various rami are indistinguishably fused. Lying dorsally of this ganglion and only imperfectly separable from it is the jugular ganglion, or ganglion of the rami cutanei dorsales (*g. X. 6*).

Remembering that these communis fibres of the vagus

supply two very different types of sensory structures peripherally (on the one hand the highly specialized taste buds and on the other hand the simplest possible free endings in the general visceral and mucous surfaces), the attempt was made to find some morphological criterion centrally for these two types of fibres. In this I was not very successful, though in the ganglia we get a suggestion which may be of some value. The anterior (cephalic) rami of the communis system undoubtedly contribute most of their fibres to taste buds and a smaller number to the undifferentiated mucosa. On the other hand, as we pass caudad the number of taste buds to be supplied diminishes, while the proportion of undifferentiated viscero-sensory fibres is greatly increased, until in the r. intestinalis and the œsophageal rami the fibres all belong to this latter category. Now, the ganglia of the glossopharyngeus and the first branchial of the vagus are composed of very large cells with medium and very small cells intermingled and occasional little nests of the smallest cells crowded very closely. As we go toward the caudal end of the ganglionic complex, we continue to find cells of the various sizes, but the smaller ones become increasingly numerous. The hypothesis suggests itself that the larger cells are related to the taste buds and the smaller ones to the visceral fibres. A careful cytological study and comparison of the ganglion cells of the several components would doubtless furnish many points of morphological value.

3.—The Cutaneous Root of the Vagus.

Peripherally the general cutaneous branches of the vagus are, as we shall see, very clearly separable from all of the other fibres of the vagus complex. On the other hand, the analysis proximally is attended with much

greater difficulty. The ganglion of this component (*jug. g.*, the jugular ganglion of Shore and Strong, not of Gaskell) is not sharply separate from the rest of the vagus ganglionic complex, yet sufficiently so to make plain that here, as in the tadpole (Strong, '95) it is the proximal portion of the ganglion which performs this function. Indeed we shall see below that the mode of origin of the rami cutanei dorsales of the vagus would of itself be sufficient to locate this ganglion quite precisely.

The root fibres of this component are so intimately intermingled with those of the large communis root, which are of nearly the same size, that I found it impossible to follow them into the brain in transverse sections. In longitudinal sections they cannot be separately followed all of the way from the ganglion to the terminal nucleus, but at the superficial origin of the vagus the small bundle of cutaneous fibres separates and, passing in between the cephalic and caudal communis roots, turns abruptly caudad into the spinal V tract.

This root entering the spinal V from the vagus I have found much larger in Haploidonotus than in Menidia and Kingsbury ('97) reports it as well developed in Amiurus, Perca, Roccus, Lepomis and Amia. Strong finds it also in the tadpole and from Kingsbury's description ('95, p. 177) it is clear that the same relation holds for Necturus, though Kingsbury was unwilling to admit the homology. The occurrence of this root is probably general throughout the Ichthyopsida.

4.—The Motor Roots of the Vagus and Glossopharyngeus.

i.—The Nucleus Ambiguus.—In the spinal cord I have described in Section 3 two nuclei which are presumably both motor, the nucleus of the ventral cornu and the para-

central nucleus at the level of the canalis centralis. The former ceases cephalad of the first spinal nerve and the latter is nearly or quite interrupted for a short distance there also, its place being taken, however, by the motor nucleus of the vagus, the nucleus ambiguus, which lies close to the floor of the fourth ventricle on each side. The rather large multipolar cells which compose this nucleus are arranged in dense rosettes, the cell group terminating very abruptly caudad. In none of the fishes which I have examined does the nucleus ambiguus gradually merge into the ventral cornu, as described by Haller ('96). Its relations are with the paracentral nucleus rather than with the ventral horn.

At the level of the exit of the most caudal fibres of the vagus nerve (663) a small compact bundle of fibres leaves this nucleus, probably deriving some fibres also from the nucleus of the opposite side, and passes to the ventral surface of the chief vagus root. The nucleus, continuing cephalad, is somewhat reduced for a short distance, but at about the level at which the most cephalic vagus fibres leave the oblongata (640) it is again greatly enlarged and at this point gives off a broad band of fibres, which is also probably re-enforced from the opposite side and which enters the vagus root and there joins the other motor root just described. The nucleus continues cephalad to 625, but crowded farther ventrad by the enlarged lobus vagi, so as to lie laterally of the fasciculus longitudinalis dorsalis. Several bands of fibres arise at intervals from the prevagal portion of the nucleus ambiguus and curve back to join the other motor fibres. This nucleus is only diagrammatically indicated in Fig. 4.

Quite separated from the nucleus ambiguus, but a very short distance cephalad is another very small nucleus of

cells of the same character lying close to the median line ventrally of the fasciculus longitudinalis dorsalis. It is partly embedded in, but chiefly dorsally of the commissura accessoria (614–611). Its fibres were not traced.

ii.—The motor vagus derives its fibres mainly from the nucleus ambiguus of the same side. It receives some fibres from the commissura accessoria which probably come in part from the nucleus ambiguus of the opposite side and in part from the fasciculus longitudinalis dorsalis.

The peripheral course of the vagus fibres from the nucleus ambiguus through the ganglionic complex can be followed only with great difficulty on account of the interlacing of the root-fibres. After emerging from the oblongata the two motor roots unite in the cephalic face of the great vagus root; they then descend to the ventral surface of the vagus ganglion and here the common trunk divides into two main branches, one directed cephalad, the other caudad. This is clearly the general course followed by most, if not all, of the fibres. Peripherally from the point of divarication of the motor fibres into the several rami they again appear sharply separated from the finer communis fibres and can thereafter be separately traced to their termini. In the ganglionic complex the motor fibres gather in bundles on the ventral surface of the ganglia. These bundles contain both coarse and deeply staining fibres and fine ones which stain feebly and which resemble communis fibres. The former are unquestionably derived from the nucleus ambiguus and can be followed to the striated musculature of the branchial apparatus, etc.; the latter are probably motor fibres derived from some other source and destined for the unstriated musculature of the œsophagus and viscera.

Kingsbury ('97, p. 5) describes in Amia fibres entering

the caudal motor root of the vagus from the ventral horn, as well as from the nucleus ambiguus. Haller ('96) describes and figures from Golgi preparation similar fibres from ventral horn cells (his ventral nucleus of the vagus) in Salmo. I have not found such fibres in Menidia, but no Golgi preparations were made and I cannot deny the possibility of their presence in small numbers. Compare, further, the last paragraph of the discussion of the m. pharyngo-clavicularis externus later in this section.

iii.—The Motor Glossopharyngeus.—From the nucleus ambiguus arise also, as in the mammals, the motor fibres of the IX nerve. These fibres arise from the most cephalic part of the nucleus and pass directly cephalad in the same position as that of their cells of origin, *i. e.*, along the lateral surface of the fasciculus longitudinalis dorsalis. This course they maintain for a considerable distance (625–600). Although closely appressed to the fasciculus longitudinalis dorsalis, these fibres can always be distinguished from it by their smaller size. There is, moreover, always a connective tissue septum between them. In this position the tract from the nucleus ambiguus divides (Fig. 18) into two distinct round bundles of which the dorsal (*IX f. l. d.*) is the larger. The ventral one alone is the motor IX root (*mot. IX*). At 600 this bundle separates to enter its nerve, but the dorsal one continues cephalad in the original position. Here the latter is joined (595) by the motor root of the VII nerve, as described below, and a little farther forward it indistinguishably fuses with that root and with the fasciculus. The motor IX root after leaving the fasciculus turns sharply caudad and laterad toward its exit, crossing the spinal V tract on the ventral side of the latter. It is joined just after its exit from the oblongata (620) by the

sensory root of the IX, which passes from the fasciculus communis to its exit dorsally of the spinal V tract.

The entire course of the motor IX fibres can be followed with precision from the nucleus of origin to the peripheral distribution. The general arrangement is essentially as described by Mayser in cyprinoids ('81). In the carp it does not form so sharp a genu and passes out dorsally of most of the fibres of the spinal V tract. Johnston ('98) describes the motor IX as arising wholly from the fasciculus longitudinalis dorsalis, evidently not having traced the fibres back to their nucleus of origin.

iv.—General Considerations.—Two classes of motor fibres emerge with the vagus of fishes, (1) coarse fibres from the nucleus ambiguus for the striated branchial musculature, (2) very fine fibres for the unstriated visceral musculature, such as the constrictor muscles of the lower part of the œsophagus, etc.

The nucleus ambiguus in these cases is the undoubted homologue of the nucleus of the same name in the mammals. The chief nucleus of the vagus in the mammals is commonly regarded as sensory (Van Gehuchten, '97, p. 481; Kölliker, '96, p. 239), yet Forel ('91) brought forth very strong evidence by v. Gudden's method tending to show that it is motor, while the descending, or spinal nucleus, which accompanies the solitary bundle, is sensory. In this he may be too extreme; nevertheless the probability remains very strong that in the mammals the chief vagus nucleus contains some motor cells. (Compare the more recent work of Van Gehuchten cited in Section 3, III).

We get a hint along the same line from the degeneration experiments of Niedzvietzky ('97, p. 534). After resection of the vagus in three adult rabbits he found cells

of the nucleus ambiguus of the same side degenerated and a smaller number of the opposite side. The "sensory nucleus of the vagus" he divides into two parts, an outer, or dorsal part, of small cells which is somewhat affected, and an inner part of larger cells, which is almost completely degenerate. It is probable that the latter is viscero-motor in function, though Niedzvietzky does not consider such a possibility. This probability is strengthened by the researches of Onuf and Collins ('98), in which it is proven experimentally that this nucleus in the cat has a large motor sympathetic element. I may add that they too regard this nucleus with its sensory and motor elements as the continuation of the "intermediate zone," or visceral centre of the spinal cord.

This intimate union of the viscero-motor and communis systems is not confined to the vagal region, but is characteristic of the entire extent of the fasciculus communis. We have a graphic illustration of this in a recent series of experiments by Langley ('98). The vagus and cervical sympathetic were cut and the central end of the vagus joined to the peripheral end of the sympathetic. The result was that "certain of the fibres of the vagus grow along the course of the cervical sympathetic and make functional connection with the cells of the superior cervical ganglion," and there was a restoration of the proper functions of the peripheral sympathetic, including some classes of fibres such as pilo-motor nerves which are not represented in the vagus root. In another experiment the proximal end of the lingual (chorda tympani) was in the same way joined to the peripheral end of the cervical sympathetic, with a similar restoration of the functions of the peripheral cervical sympathetic. These interesting experiments go to show that sympathetic motor (pre-gan-

glionic) fibres go out with all of the roots from the communis system and that these fibres are so similar to the corresponding fibres which go out with the first spinal roots that they are capable of functional substitution for them.

In a preceding section we suggested the theory that in the oblongata the unspecialized visceral sensory centre corresponding to that of the spinal cord has had differentiated from it the special sensory system for the terminal buds, a sensory system which is not represented in the spinal cord. So also in the head there seems to have been a special differentiation of the viscero-motor system (nucleus ambiguus, motor VII and motor V) co-ordinate with the development of the branchial motor apparatus, which we know to be derived, not from the somites, but from the splanchnic musculature.

The suggestion made by Cole in a recent paper ('98, p. 233, foot note) is interesting in this connection: "A possible explanation of the vagus, I think, is that the branchial nerves are secondarily sympathetic, *i. e.*, in function only, whilst the visceral nerve is primarily sympathetic, *i. e.*, represents a modified portion of the sympathetic, and thus both physiologically and morphologically belongs to that system. Its connection with the vagus is thus a 'blind' and of precisely the same significance as the connection of the sympathetic with the trigeminus and facialis." The viscero-motor fibres of the trunk belong to Langley's type of pre-ganglionic fibres, *i. e.*, they terminate in sympathetic ganglia and reach their appropriate visceral muscles only through the mediation of sympathetic fibres. The same may hold true of the viscero-motor fibres of small calibre which go out with the vagus, but it is certainly not true of the motor fibres of large size

which go out from the n. ambiguus to the striated branchial musculature. If these latter fibres have been derived from the pre-ganglionic system, they have certainly suffered a most remarkable metamorphosis. If, as implied in Cole's remark above, they have not been thus derived, we should find it difficult to find any homologue for them in the trunk and we may be led to assume that they belong primarily to the branchial region, for several lines of current research seem to hint at the possibility that this region may be older than the trunk after all.

5.—The Root of the R. Lateralis Vagi.

The root of the lateral line nerve, after separating from the lateral aspect of the tuberculum acusticum (610) and emerging from the oblongata immediately dorsally of the origin of the IX nerve, as it passes caudad, sinks down until it comes to rest upon the dorsal surface of the ramulus ampullæ posterioris (Fig. 17). At this level it begins to be crowded laterally by the emerging vagus root, upon the ectal surface of which it is closely appressed without, however, anastomosing with it at all. It passes through the same foramen as the vagus. The auditory ramulus referred to almost immediately separates and turns laterad to the proper auditory organ, without any interchange of fibres with the lateralis root.

The lateral line nerve at its origin is not composed exclusively of the characteristic very large and heavily myelinated fibres, but there are numerous medium-sized fibres. These may be diffusely scattered through the trunk or they may be gathered into rather compact bundles, whose positions and relations, however, vary in different specimens. They can be followed into the ganglion of the lateral line nerve. Their peripheral relations and

central connections are unknown. The largest root fibres are fully 12 micra in diameter and the average size of all of the fibres is about 8 micra, while the smallest fibres are scarcely 3 micra.

About two-thirds of the distance between the exit of the r. lateralis and its ganglion it receives a small bundle of fibres (about 20 in number) from the IX root. This little twig is of great interest and has been carefully worked out in a number of specimens. The fibres are of small or medium size, considerably smaller than the lateralis fibres, but larger than the average communis fibres, the largest ones being 3 or 4 micra in diameter. After leaving the IX root, they pass dorsad and caudad along the mesal face of the ramulus ampullæ posterioris and close to the medulla oblongata to enter the ventro-lateral surface of the r. lateralis. On the opposite side of this specimen and in two others they run first up the mesal side of the r. lateralis, then laterally around the dorsal side of this ramus, finally to enter it in the same position as before described.

Neither these nor the other fine fibres of the lateralis root could be separately traced through the ganglion. This ganglion contains medium-sized ganglion cells, much larger than those of the VIII ganglion, but smaller than the largest of the vagus ganglion. There are a few larger cells among them. A considerable number of finer fibres leave the lateralis ganglion which are presumably related to those which enter it. Their further courses will be described below.

II.—The N. Glossopharyngeus.

The organization of the IX nerve is exceptionally simple. Its peripheral area is limited to one demibranch. It lacks the r. supra-temporalis, which is present in

Chimæra, in most selachians, in ganoids, siluroids and some other fishes, and accordingly it receives neither lateralis nor general cutaneous fibres. The r. pre-trematicus is also totally wanting. The ramus post-trematicus is, however, identical with the corresponding branches of the several branchial trunks of the vagus. The nomenclature employed for the branchial musculature and for the cranial musculature in general is that of Vetter ('78).

The motor IX fibres from the n. ambiguus run along the ventral side of the root and at the ganglion they separate slightly from the latter ventrally, to rejoin the nerve beyond. The motor component in this region just below the ganglion gives off fibres for the first m. levator arcus branchii internus (*r. l. a. i.*). Just beyond the ganglion a very minute pharyngeal ramus (*ph. IX*) is given off from the fine-fibred component for the mucosa of the roof of the mouth above the first gill, then a minute motor twig for the first levator arcus branchii externus (*r. l. a. e.*).

All of the remaining fibres enter the first gill, just before entering which they divide into two nearly equal ramuli, a dorsal and a ventral. The latter contains all of the remaining motor fibres and a somewhat larger number of fine communis fibres, passes down the outer or convex surface of the ceratobranchial bone and supplies the muscles of the gill filaments and their mucous surface. It lies dorsally of the first demibranch and probably distributes most of its fibres to it. The dorsal ramulus pursues a course parallel to the last, but on the opposite, or concave side of the ceratobranchial bar, distributing to the large taste buds with which this dorsal surface of the gill is plentifully supplied and to the long gill rakers. Probably many of its fibres supply the general mucous surface,

though it is certain that most of them go to taste buds. The sensory fibres of the ventral ramulus, on the other hand, apparently do not go to taste buds as a rule. After the first gill has joined the isthmus the dorsal ramulus continues for a short distance to supply taste buds in the floor of the mouth; the sensory fibres of the ventral ramulus, after joining the pre-trematic ramus of the first branchialis vagi, pass to similar taste buds nearer the median line than the last, while the remaining motor fibres separate for the m. obliquus ventralis of this gill. On Fig. 3 none of the branchial ramuli of the IX and X nerves are plotted for their full length except the dorsal one of the IX nerve.

The absence of the r. pre-trematicus IX (r. hyoideus) is, I think, to be accounted for by the peculiar relations of the pseudobranch. This structure is very large indeed and occupies the whole region above and in front of the first gill cleft. It, however, belongs to the region of the facial; the peripheral region pertaining to the hyoidean branch of the IX nerve having been thus crowded out, the loss of the nerve naturally follows. Stannius reports the absence of the r. pre-trematicus IX in Esox and Silurus and its great reduction in Belone ('49, p. 76). The same cause has operated also to reduce the r. pharyngeus. Cole ('98a, p. 145) has shown that in the fishes, as in the mammals, this ramus usually joins the visceral portion of the facialis and thus is a true Jacobson's anastomosis. The commissure from the IX to the VII in Menidia is, however, purely sympathetic (see Section 8).

III.—First Truncus Branchialis Vagi.

The origin and course of this nerve from its ganglion are strictly comparable with those of the glossopharyngeus. There is given off first the minute motor twig for

the second m. levator arcus branchii externus (*2. l. a. e.*) and then the truncus divides into pre-trematic and post-trematic rami. The former is quite small. It gives off immediately a pharyngeal ramus (communis fibres) for the roof of the mouth adjacent, which, though slightly larger than that of the IX nerve, is nevertheless inconsiderable.

The pre-trematic ramus, upon entering the first gill, takes its position on the outer or convex surface of the ceratobranchial bar just above the attachment of the second demibranch to the bar, where it may be followed to the base of the gill. Here it joins the ventral ramulus of the post-trematic IX and can no longer be separately traced. This anastomosis has been found only in the first gill. If it occurs at all in the others, it can involve only the ultimate terminal fibrils of the pre-branchial nerves, as careful examination has failed to demonstrate it. The other pre-trematic branches are smaller and can be traced into their respective gills a much shorter distance than this one.

The post-trematic ramus pursues a course in the second gill which is substantially similar to that of the IX in the first gill. The most important difference (aside from the apparent absence of the anastomosis of the ventral ramulus with the second pre-branchial) is correlated with the fact that the long gill rakers of the first gill are wanting in the second, their place being taken by numerous teeth born on the dorsal side. To these teeth and the large taste buds among them the dorsal ramulus is distributed, as well as to the taste buds of the floor of the pharynx near the base of the gill. The dorsal and ventral ramuli are about equal in size.

The ganglion of the first branchial nerve of the vagus is

more distinct than that of any of the other branchials, and this seems to be the rule among the teleosts. Stannius states that in Clupea and Alosa this ganglion is fused with the IX ganglion. He does not mention the division of the post-trematic nerve into dorsal and ventral ramuli. Baudelot, however, describes ('83) a somewhat similar condition in the carp, though there it is the pre-trematic ramus which is double, the dorsal ramulus being much larger than the ventral.

IV.—Second Truncus Branchialis Vagi.

This division of the vagus gives rise to four branches for the roof of the mouth, two motor and two mixed. First, there are the two purely motor twigs, one, very minute, for the third levator arcus branchii externus (*3. l. a. e.*), the other for the single large m. branchialis obliquus superior (*m. b. o. s.*). The third branch leaves the cephalic face of the post-trematic ramus and passes partly through, partly laterally of the second m. levator arcus branchii internus (*2. l. a. i.*). In its passage through this muscle it forms an intricate plexus and doubtless innervates it, though most of the fibres emerge and anastomose with the more lateral fibres, finally to enter the dentary canal in the upper pharyngeal bones (pharyngo-branchials). The remaining fibres of this pharyngeal nerve distribute to the mucus membrane of the lateral part of the pharynx (sensory buds and glands).

Regarding the levator arcus branchii internus muscles, there seem to be but two muscles in this series. The second one is much larger and longer than the first one. It originates from the skull far cephalad at the level of the exit of the third nerve and passes back almost horizontally to its insertion on the second pharyngo-branchial bone, so that it really serves as a protractor of these bones. The first levator arcus branchii internus arises from the skull much farther caudad

and inserts into the first pharyngo-branchial bone, thus passing more obliquely and serving as a levator as well as a protractor. It is innervated from the IX nerve.

The fourth pharyngeal branch (*ph. X. 2.*) arises directly from the ganglion immediately behind the origin of the truncus. It is mixed, containing both communis and motor fibres. It passes directly ventrad and mesad to the roof of the pharynx near the mid-line, passing along the mesal face of the very strong m. obliquus dorsalis superior and receives an anastomosing branch from the pharyngeal branch of the third branchialis vagi, as described below. In the roof of the mouth it turns cephalad, supplying richly the racemose glands of this region, the taste buds at their mouths and the small striated muscles at their bases.

The pre-trematic and post-trematic rami are as in the preceding case. (Fig. 1).

V.—Third Truncus Branchialis Vagi.

This trunk divides into pre- and post-trematic branches immediately upon leaving the ganglion. Each of these branches gives off a large pharyngeal ramus, the aggregate number of these latter fibres being much greater than in any of the preceding branchial nerves. These pharyngeal rami (*ph. X. 3*) pass ventrad to the superior pharyngeal teeth; they probably also supply taste buds adjacent. They anastomose freely with each other and with the most caudal pharyngeal ramus of the second division, so that the innervation of the roof of the pharynx in this whole region is very intricate. These plexiform anastomoses are not entered upon the plots. There are also two motor rami. One passes directly from the ganglion to the fourth m. levator arcus branchii externus

(*4. l. a. e.*), the other passes from the post-trematic nerve to the m. obliquus dorsalis (*m. o. d.*).

The pre- and post-trematic nerves are as before, save that at the base of their gill (the fourth) the remaining motor fibres of the ventral ramulus supply the m. transversus ventralis (one of the interarcuales ventrales) instead of the m. obliquus ventralis, which does not appear in this gill. The terminal sensory fibres of this ramulus supply the enormous taste buds near the median line of the floor of the mouth, those of the dorsal ramulus similar buds farther laterally, as in the other gills.

VI.—Fourth Truncus Branchialis Vagi.

The fourth branchial (*t. X. 4.*) separates from the general vagus complex farther caudad than any of its other rami. As in the preceding cases, it draws off both coarse and fine fibres. As it arches around dorsally from the mesal to the lateral aspect of the m. obliquus dorsalis posterior (of Vetter) it contributes most and probably all of its coarse fibres for the innervation of this muscle (*m. o. d. p.*).

This muscle, passing from the fourth gill bar to the caudal edge of the lower pharyngeal bone and classed as one of the interarcuales dorsales, is described by Vetter ('78, p. 509) as peculiar to Esox. The relations in Menidia conform in general to his description, though the muscle here seems to be larger and to arise mainly from the fourth epibranchial instead of from the ceratobranchial, as in Esox.

The pre- and post-branchial rami are both very small. The former passes to the fourth gill in the manner typical for the other gills: the latter is very minute and composed exclusively of fine, *i. e.*, sensory fibres. There being no gill behind the fifth gill cleft, it distributes to the general mucosa dorsally of the beginning of the œsophagus and

about the caudal end of the inferior pharyngeal bone. There are no obvious taste buds in this region. If present, they are very small.

VII.—Other Visceral Rami of the Vagus.

Under this head I shall describe motor and sensory rami of the vagus which distribute caudad of those visceral rami which accompany the branchial nerves. The ganglion of this division cannot be separated from that of the fourth branchial and only imperfectly from the jugular, or general cutaneous ganglion (*jug. g.*). There are few, if any, taste buds in the area supplied by it.

1.—Ramus Intestinalis.

Most of the fibres of this division are destined for the r. intestinalis or the equally large r. œsophageus. The former (*r. intest. X.*) runs caudad between the kidney and the dorsal wall of the œsophagus, giving off fibres to the latter from time to time and anastomosing at intervals with the main sympathetic chain of the trunk which lies farther dorsad. Its farther course was not traced.

2.—Œsophageal Rami.

At the point where the intestinal ramus separates from the branchial complex (730) several very large trunks of apparently the same nature (*r. œs.*) turn directly ventrad to the œsophagus, which is just closing off from the pharynx at this level. These fibres supply the strong œsophageal muscles and probably also the epithelium, which is here very glandular. No taste buds are present. The innervation of this region is surprisingly rich and most of the fibres are apparently motor. Both the circular and longitudinal muscular fibres of the œsophagus

which are supplied by these nerves are all or nearly all striated. In the plots I have not distinguished these fine viscero-motor fibres from the sensory fibres which they accompany because of the impossibility of analyzing them here or of tracing them to their nucleus of origin. In Fig. 4. the œsophageal and infra-pharyngeal rami have been slightly schematized; really they consist of several trunks with an elaborate plexiform anastomosis.

3.—Ramus Cardiacus.

As the œsophageal fibres curve ventrally around the œsophagus, a small bundle separates and accompanies the adjacent precaval vein to the heart (*r. car.*) This is the cardiac nerve. Its fibres are partly very fine and partly of medium size and heavily medullated.

4.—Pharyngeal Rami.

Of the sensory fibres, a small twig supplies the most caudal superior pharyngeal teeth (*ph. d.*), while several considerably larger nerves enter the caudal edge of the inferior pharyngeal bone for its teeth (*ph. v.*)

5.—Branchio-Motor Rami.

Besides the fibres above referred to, I enumerate with this division the following coarse fibred motor nerves for the branchial musculature. They can easily be traced back to the common motor bundle of the vagus and so with tolerable certainty to the nucleus ambiguus. As indicated on Fig. 4, the following motor nerves are given off:

i.—Ramus for m. transversus dorsalis (*m. tr. d.*)—This muscle runs from side to side between the two superior pharyngeal bones.

ii.—Ramus for m. retractor arcus branchii dorsalis (*m. r. d.*), a muscle which runs from the second and third

vertebræ to the superior pharyngeal bone (Vetter, '78, p. 511).

iii.—Ramus for m. pharyngeus transversus (*m. ph. t.*)—This is a large stout muscle extending between the two inferior pharyngeal bones. It is incompletely divided into two parts, a large ventral part which is supplied by a small number of very coarse and heavily myelinated fibres, like those for the other branchial muscles which can be traced back into the common motor component, and a smaller dorsal part which is dorsally confluent with the general constrictor muscles of the œsophagus and like them is supplied by many very fine fibres whose origin could not be traced. The muscular fibres of the ventral part are very large and thick, those of the dorsal part smaller, but not so small as those of the proper constrictor of the œsophagus.

iv.—Ramus for m. pharyngo-clavicularis internus (*m. ph. c. i.*)—This large muscle has its origin on the dorsal edge of the cleithrum near its ventral end and passes almost directly dorsad to its insertion along the lower edge of the caudal part of the inferior pharyngeal bone.

v.—Ramus for m. pharyngo-clavicularis externus (*m. ph. c. e.*).—This is a short thick muscle which has a tendinous origin from the inner face of the cleithrum farther caudad and much farther dorsad than the last. Its fibres are directed mesad, cephalad and slightly ventrad and cross those of the m. pharyngo-clavicularis internus at about a right angle, being closely appressed to the caudal face of the latter muscle near its insertion. Their insertion is tendinous upon the extreme ventro-caudal tip of the inferior pharyngeal bone ventrally of the insertion of the m. pharyngo-clavicularis internus and laterally of the caudal part of that of the m. pharyngeus transversus.

The motor fibres for the last two muscles leave the œsophageal complex near the point where the fourth branchial trunk separates, encircle the œsophagus, then divide and enter their respective muscles from behind and can easily be traced to their ultimate ramifications within these muscles.

This account confirms the general statement of Stannius ('49, p. 90). Wright ('84, p. 371) states that the pharyngo-claviculares are supplied by the first spinal nerve in Amiurus, and Harrison ('95) makes the same statement for Salmo. Vetter ('78, p. 524) states that they are supplied by the vagus in Esox; in Perca he demonstrated the innervation of the internal muscle from the vagus, but in Cyprinus he found that the ventral nerve formed from the first two spinal nerves (XII nerve of authors, r. cervicalis of Fürbringer) runs along the hinder edge of the m. pharyngo-clavicularis internus and he thought that the hinder part of this muscle receives some fibres from that source. In selachians and chimæroids these muscles are supplied by the spinals (Fürbringer, '97). In Amia (Allis, '97, p. 697) they are supplied from the vagus essentially as in Menidia, though in that case their nerve runs out with the ramus post-trematicus n. vagi quarti. Allis corrects McMurrich's statement ('85, p. 138) that the claviculares in Amia are supplied by the first spinal. The nerve (which really is the fourth spino-occipital nerve) merely traverses the muscle.

Fürbringer ('97, p. 469) describes the external and internal pharyngo-claviculares (his cleido-branchialis 5, external and internal) as innervated from the spinal nerves in Esox, Gadus and Caranax, and he considers that Stannius and Vetter are in error when they describe them as supplied by the vagus. In this it would appear as if Fürbringer were unduly influenced by theoretical considerations. Regarding the cleido-branchiales as somatic muscles, it accords ill with his scheme of somatic and visceral nerves to find these muscles innervated from the

paleo-cranial (and hence visceral) motor rami of the vagus (compare the discussion of Fürbringer's theories in topic V of Section 4).

As a matter of fact it is probably quite impossible in some cases to determine by dissection which of these nerves supplies the pharyngo-claviculares or whether both sometimes participate, as Vetter supposed. For example, in Menidia the r. cervicalis of the first spinal nerve, as we have seen, runs down immediately behind both of these muscles and closely applied to their caudal surfaces along their entire extent. The relations of this nerve to the internal muscle are especially intimate and while descending along its caudal face several small cutaneous twigs are given off which pass laterally along the surface of the muscle on their way to the skin. These twigs are very minute and their courses tortuous, and it is doubtful whether even in a large fish they could be dissected out to their terminations, though their whole courses can be easily followed in the sections. It is probably these fibres which Vetter supposed innervated the hinder part of the muscle and he was mistaken in regarding them as motor, but correct in his further supposition that, aside from these fibres, a branch of the vagus which he had overlooked furnishes the main innervation for these muscles.

In the case of Menidia there is no room for doubt that the relations are as above described. My sections are perfect and absolutely free from ambiguity. Of course it does not follow that the same conditions prevail in all teleosts. If it is true that these muscles are innervated from the first spinals in Amiurus (Wright) and in the lower fishes (Fürbringer), this raises the question whether the muscles so named are homologous in Menidia and Amiurus and whether the pharyngo-claviculares of the teleosts are homologous with the coraco-branchiales of selachians and ganoids, as Fürbringer assumes to be the case.

An embryological examination may be necessary to decide this matter, which is, however, of some theoretical

importance, as mentioned above under the first spinal nerve; for if these are somatic muscles, as Fürbringer maintains, their innervation from the vagus can be explained only upon the assumption that either (1) one or more neo-cranial segments have been secondarily incorporated into the vagus nerve, or (2) that a paleo-cranial nerve may secondarily come into relation with a somatic muscle belonging to a neo-cranial segment. Both of these assumptions are denied by Fürbringer. It should be mentioned that there is one further possibility, viz., that these muscles may be innervated by somatic motor fibres from the ventral horn which go out with the vagus roots. Fürbringer does not admit this possibility either, and I have not found such fibres in Menidia, but they are described for Salmo (Haller, '96) and for Amia (Kingsbury, '97).

VIII.—Rami Cutanei Dorsales Vagi.

These comprise those general cutaneous fibres which are included with the vagus. Three very delicate nerves arise from the jugular ganglion, which lies proximally and just dorsally of the ganglion of the intestinal and fourth branchial rami and only imperfectly separable from it. These nerves pursue devious courses to the skin of the opercular and supra-opercular regions. Two of these arise from the caudal surface of the ganglion. One, directed caudad (*cut. X. 3*), is accompanied for a short distance by the motor ramus for the trapezius muscle described below. It then turns dorsad between the latter muscle and the dorsal musculature, to reach the skin at the caudal edge of the attachment of the operculum. Both the outer and the inner surfaces of the operculum at this point are supplied.

Arising with the last, but abruptly turning cephalad, is a second slender cutaneous twig distributing farther dor-

sally (*cut. X. 2*). It goes cephalad and dorsad under the mucous lining of the pharynx at the extreme mesal angle of the gill chamber. Under the caudal edge of the parotic process of the skull (640) it turns laterad and somewhat caudad and dorsad and distributes in numerous small branches to the skin adjacent to the lateral line canal caudad of this point.

From the cephalic face of the jugular ganglion the other cutaneous ramus (r. opercularis vagi) arises by two roots, both directed cephalad, one going ventrally, the other dorsally of the main vagus root, just cephalad of which (660) they unite and, turning dorsad, continue cephalad (*r. op. X.*). They appear to be composed exclusively of cutaneous fibres, but as these roots are very minute and their courses sinuous, it is impossible to be certain that at their emergence from the general vagus root they do not carry with them some communis fibres also. Some points in their distribution suggest that they do, though most of the fibres are clearly cutaneous, and as such all have been entered upon the plot (Fig. 4).

From the vagus root this twig goes cephalad and slightly dorsad and laterad under the parotic process, and at the origin of the m. adductor operculi runs laterally between this muscle and the parotic process to a position on the lateral face of the muscle between it and the levator operculi (610). In this intermuscular space it continues cephalad and ventrad and crosses the deep, or motor, ramus opercularis VII. It passes externally to the latter nerve and in contact with it, but there is no interchange of fibres, as Baudelot ('83) states is the case in the cyprinoids.

Having passed below the ventral edge of the m. levator operculi, it comes to lie between the m. adductor operculi

and the m. dilator operculi and here it breaks up into several branches which spread out over the inner surface of the latter muscle, (1) one extends cephalad and dorsad, (2) one caudad and dorsad, (3) one caudad and ventrad, and (4) one directly ventrad. Of these, all but the last are undoubtedly cutaneous nerves. The first one (*op. X. 1*) runs between the m. levator operculi and the m. dilator operculi to the skin under the lateral line canal; the second one (*op. X. 2*) can be traced along the inner surface of the m. dilator operculi for almost its entire length (back to 700), from time to time giving off twigs which break through this muscle to the skin of the dorsal part of the operculum. It innervates the cutaneous area lying between those supplied by the two caudal cutaneous branches (*cut. X. 2* and *cut. X. 3*).

The third branch (*op. X. 3*) also runs back along the inner surface of the same muscle, just dorsally of the upper edge of the opercular bone and farther back along the outer side of the dorsal surface of this bone between it and the m. dilator operculi. It can be followed nearly to the caudal end of this muscle (720) and finally passes through it at its insertion on the opercular bone to end in the skin of the dorsal part of the operculum. The area supplied is ventral to that supplied by the *r. cut. X. 3*. This is an exceedingly delicate nerve, containing only about ten fibres in all, and could not be traced with certainty even sections, but for the fact that the fibres are heavily myelinated, being nearly as large as typical motor fibres. Their cutaneous nature is, however, beyond question.

It is probably the intimate relation of the three cutaneous branches just noted to the m. dilator operculi which led Baudelot ('83) to describe branches of the oper-

cular ramus of the vagus as innervating the opercular muscles.

The fourth branch of the the r. opercularis vagi (*op. X. 4*), composed of very fine fibres, passes directly ventrad after separating from the third branch and joins the third branch of the r. opercularis superficialis VII (*op. s. VII. 3*) just above the dorsal edge of the opercular bone. The combined trunk now passes down the inner face of that bone and supplies the mucous membrane of the dorsal part of the lining of the operculum.

Baudelot ('83, p. 132) says of this nerve in the carp: "*Quant à la branche operculaire du pneumogastrique, elle descend jusqu'au bord inférieur de l'opercule en envoyant sur son trajet des filets à la peau: il est possible qu' elle s'anastomose à sa terminaison avec la branche operculaire superficielle*" [VII].

Guitel ('91) finds in Lophius that this nerve anastomoses with both the superficial and the deep opercular rami of the facial, and that the nerve for the mucous lining of the branchiostegal membrane comes from the latter anastomosis, instead of the former, as I find it in Menidia.

These cutaneous vagal fibres are of special interest. Their true relations seem not to have been clearly recognized by any of the older writers. Shore ('89) found in Raja a cutaneous branch in this position which he calls r. auricularis and regards as general cutaneous. Nevertheless it is not quite clear from his description (especially as he describes it as coarse fibred) that this may not be the r. supratemporalis vagi (lateralis fibres), which Ewart and Mitchell ('91) describe as arising in about the same position. It is probable that his cutaneous ramus contains both of these elements, as in Raja there is no r. supratemporalis IX for the canal organs (Stannius, p. 79, Ewart and Mitchell, '91).

Strong was the first to contrast clearly the r. supratemporalis (a lateral line nerve) and the r. cutaneous dorsalis

(= r. auricularis vagi), and indeed has predicted for fishes just the arrangement which we have here ('95, p. 155): "The question arises, then, as to what is the homologue in *lower* forms of the R. auricularis vagi of the tadpole. I may here simply say that I believe that future investigation will bring out more clearly a system of general cutaneous branches in this region coexistent with the lateral line nerves."

Allis' account ('97, p. 689) of these nerves in Amia obviously meets these conditions also, though he does not seem to have interpreted his facts in just this way. His first branch, or r. supratemporalis, of the vagus has two roots. One clearly comes from the nervus lineæ lateralis and was traced to lateral line organs. The other arises from an intra-cranial ganglion which corresponds perfectly to our jugular ganglion. These fibres distribute to the skin of the top of the head and the opercular region. Allis repeatedly emphasizes the fact that this nerve distributes to regions where terminal buds abound, and, though he is not explicit on this point, the inference is that he regards it as wholly composed of communis fibres. That it is largely so composed is probable from his description, but it is equally probable that some of these fibres are of general cutaneous character and comprise the r. cutaneous dorsalis.

It is evident from the descriptions of Stannius and Baudelot that the r. supratemporalis vagi (lateralis fibres) and the proper r. cutaneous dorsalis vagi are in some teleosts fused for the first part of their course, just as in general we find both lateralis and general cutaneous fibres in the analogous r. oticus. In the cyprinoids, for example (Baudelot, '83, p. 133), we have this nerve arising by two roots furnished with separate ganglia and giving rise to opercular rami, anastomosis with r. recurrens V [VII] (=r. lateralis accessorius) and doubtless also lateral line organs, though the latter are not mentioned.

Stannius describes (p. 85) an intra-cranial branch of the vagus in several fishes which doubtless corresponds sometimes to the r. supratemporalis; yet as it contains, in some

of the cases at least, general cutaneous fibres, his account may be introduced here. He finds it to contain both coarse and fine fibres, from the lateralis root and the chief vagus root respectively, but no motor fibres, as shown by the negative results of stimulation. In this latter point he differs from Baudelot, as we saw above. Stannius adds that the intra-cranial branch is absent in Scomber, Pleuronectes, Rhombus, Salmo, Coregonus, Ammodytes, Clupea, Silurus, Spinax and Raja. It is present in Perca, Acerina, Cottus, Trigla, Barbus, Caranax, Zoarces, Cyclopterus, Labrus, Belone, Gadus, Merlangus, Raniceps, Lota, Cyprinus, Abramis, Gobio, Tincta, Esox, Acipenser and Anguilla. Now Perca, Acerina, Cottus, Zoarces, Cyclopterus, Labrus, Belone, Gadus and Anguilla possess a r. recurrens V [VII] and this intra-cranial branch anastomoses with it. Stannius states that all forms which have a r. recurrens V [VII] also possess this intra-cranial branch, with the single exception of Silurus. He describes the intra-cranial branch also in those fishes which have a dorsal intra-cranial or cranial branch of the V [VII] nerve, with which it usually anastomoses, as in most cyprinoids. Or it may pursue an independent course to the skin, as in Barbus and Caranax trachurus, or to the meninges, as in Esox. It occurs, finally, in cases where there is no recurrens V [VII] and distributes mainly within the cranium, as in Trigla and Acipenser.

In the species enumerated by Stannius as lacking the r. cutaneous dorsalis it will probably be found that the nerves are so small and sinuous as to have eluded discovery. The anastomosis with the r. recurrens V [VII], or r. lateralis accessorius, described by Stannius and Baudelot probably pertains to a communis component of the cutaneous nerves. A similar anastomosis occurs in Menidia, though in this case the communis fibres go out with the r. supratemporalis rather than the cutaneous rami. See the account of the r. supratemporalis vagi below for further discussion of the morphology of these nerves, and Section 12 for discussion of the anastomoses with the r. lateralis accessorius.

IX.—Ramus for M. Trapezius (Accessorius).

A small motor twig (*XI*) which leaves the vagus complex at the ventral edge of the jugular ganglion is worthy of separate treatment. It passes caudad for a short distance in company with the r. *cut. X. 3* and supplies the m. trapezius. It is a very small nerve, containing only from ten to twenty fibres, a much smaller number than the cutaneous nerve which it accompanies. It is very sharply separable from the latter by reason of the greater size of its fibres. The sections are quite unambiguous and leave no doubt that this nerve actually terminates in the m. trapezius and does not merely pass through it. The accompanying cutaneous fibres do not enter the muscle, but pass to the skin mesally of it. The participation of spinal nerves in the innervation of this muscle has been definitely excluded. There are no spinal nerves in its vicinity and the entire course of the muscle has been examined microscopically without revealing any spinal fibres within or near it. As to the central relations of this nerve, we can only say that its fibres arise from the common motor root of the vagus.

The trapezius muscle arises from the ventral surface of the parotic process of the skull (Parker's term) and extends back as a well defined round bundle at the lateral edge of the dorso-lateral musculature to its insertion on the dorsal part of the cleithrum. The dorso-lateral musculature of the corresponding region arises from the dorsal surface of the parotic process and from the skull dorsad and cephalad of it.

Vetter ('78, pp. 526 and 541) states that an independent trapezius muscle from the cranium to any part of the shoulder girdle is absent in the bony fishes and that the

dorsal musculature running from the skull to the extra-scapular (post-temporal) and supra-clavicular bones and supplied by the first spinal nerves is not homologous with the trapezius of selachians and Chimæra, but is a new structure in the teleosts. On the latter point he is doubtless correct, *i. e.*, so far as the proper dorso-lateral musculature is concerned; but in Menidia the trapezius muscle is present in the typical form in addition and receives the typical innervation, so that Vetter's first statement will require modification. The "trapezius" muscle described by McMurrich ('84) in Amiurus is stated to be innervated from the first spinal, so that it will fall under Vetter's rule. In Lophius, however, the case is apparently the same as in Menidia, for here the "humero-mastoid" muscle is supplied by the vagus (Guitel, '91).

Vetter regards the m. trapezius of selachians and chimæroids as derived from the superficial "Ringsmusculatur," or constrictor system. Its innervation from the vagus accords with this.

Max Fürbringer ('97, p. 374) verifies the accounts of Rohon, Duméril and Owen that the caudal ascending motor vagus root in Hexanchus is the accessorius Willisii. He has traced its connection in this species with the m. trapezius, which is especially well developed, and thus demonstrates its homology with the accessorius. He controverts the view of Stöhr, Wiedersheim and others that the XI is a descendant of spinal nerves; but regards the vago-accessorius as indigenous to the head, "a primordial cranial nerve which has nothing to do with spinal nerves." This seems probable anatomically but requires embryological confirmation.

X.—The Ramus Lateralis Vagi.

The ramus lateralis at its ganglion turns abruptly outward under the dorsal musculature and soon turns caudad. Its branches will be described from before backward.

1.—Ramus Supratemporalis Vagi.

This nerve arises from the dorsal surface of the lateralis ganglion and runs cephalad under the dorsal musculature and external to the auditory capsule. About two-thirds of its fibres are of the coarse lateralis variety, the remainder being finer fibres probably in part derived from the anastomosing communis root from the IX nerve, and representing the vagal root of the r. lateralis accessorius. About .3 mm. from the ganglion it gives off a branch which passes dorsad and caudad to the single canal organ of the occipital commissure, which lies in the same transverse plane as the lateralis ganglion. This branch, though mainly coarse-fibred, draws off a few fine fibres (*a. r. VII. 2* of Fig. 4) which, instead of turning back to the canal organ, go directly dorsad to the skin, under which they pass dorsad and then cephalad to join the r. lateralis accessorius near the median line (600, Fig. 3).

The remainder of the r. supra-temporalis (partly coarse and partly fine fibres) continues cephalad along the dorsal surface of the squamosal bone, being crowded laterally and dorsally by the projecting ear capsule. At 620 the coarse fibres separate and enter the canal organ (Fig. 5, *m. 1.*) of the main canal between the mandibular and infra-orbital canals. The finer fibres continue cephalad for a time along the squamosal bone just dorsally of the bony canal, gradually passing laterally to the skin under which they continue cephalad to 550. Here they divide, the larger portion turning directly laterally to supply a

large naked cutaneous sense organ which lies just above and in front of the pore at the union of the supra- and infra-orbital canals. The smaller portion turns abruptly dorsad under the skin (*a. r. VII. 1* of Fig. 4) and joins the main trunk of the r. lateralis accessorius near the median line, as shown on Fig. 3. The branch which supplies the sense organ contains slightly larger fibres than the one which joins the r. accessorius, many of the former being 3 micra in diameter, while the latter rarely exceed 2 micra. This organ is, I think, a terminal bud, though it may be a free organ of the lateral line series. Another similar organ was found on the opposite side of the specimen plotted and in a second specimen, lying less than 1 mm. cephalad and somewhat laterally of the upper end of the occipital commissure of the lateral line canal. Its innervation could not be determined.

The very brief reference to the r. supra-temporalis in my Anzieger preliminary ('97, p. 427) has given rise to a misunderstanding. Cole (98a, p. 185) interpreted this as implying that the r. supra-temporalis for the canal organs *m. 2* and *m. 4* is a r. supra-temporalis IX. As a matter of fact the glossopharyngeus has no r. supra-temporalis in Menidia.

2.—*The First Lateral Ramulus.*

The first nerve given off from the r. lateralis behind its ganglion (*r. l. 1*, Figs. 3 and 5) supplies the first organ of the lateral series behind the occipital commissure, viz., the single organ contained in the post-occipital canal. It contains a few fine fibres among the coarse ones. The latter are not as large as some lateralis fibres, being from 5 to 7 micra. These supply the canal organ, while the fine ones, which are about 3 micra in diameter, separate

from the ramulus in about the middle of its course and turn caudad, anastomosing in the dorsal muscle with the branch of the next ramulus, about to be described, which joins the r. accessorius lateralis (see Fig. 4).

3.—The Second Lateral Ramulus.

This follows close upon the preceding ramulus, and like it contains both coarse and fine fibres. It runs dorsad and in the dorsal muscles divides at once, one portion turning cephalad, the other caudad. At the point of division it effects an anastomosis with the ramus medius *c* of the first spinal nerve, as described in the account of that nerve, some of the latter fibres going out to the skin in the vicinity of the lateral line organ supplied by this ramulus. The fibres of both of these divisions are chiefly of medium size, those for the lateral line organ being from 4 to 6 micra, the others from 1 to 4. The portion running cephalad receives the minute anastomosing twig from the first ramulus, mentioned above, and then, while still within the dorsal musculature, divides. One twig, of fine fibres, turns dorsad and caudad to a naked cutaneous sense organ resembling the one innervated from the r. supratemporalis and situated a short distance dorsad and cephalad of the open caudal end of the post-occipital lateral line canal (735). The other twig (*a. r. VII. 3*, Fig. 4) runs dorsad, and then caudad under the skin and joins the r. lateralis accessorius (830, Fig. 3). These fibres, though slightly coarser than those of the other twig, are still finer than any proper lateralis fibres.

The portion of the second ramulus which is directed caudad, also divides into two twigs. One of these supplies the second post-occipital organ of the lateral line, which is the first free organ of the trunk. The other

twig goes dorsad through the muscle to the skin under which it continues caudad, at the same time turning toward the median line. On account of imperfections in the sections it could not be traced to its termination. It seems strictly analogous with the other twigs which join the r. lateralis accessorius, and I have no doubt that this is its destination.

It is probable that there are other cutaneous sense organs in this region supplied from these anastomosing twigs, which I have overlooked. Indeed in the younger specimens examined these have been found, though their nerve supply has not been traced.

4.—The Third to Fifth Lateral Ramuli.

The ramus lateralis was not traced back beyond the fifth ramulus. The last three ramuli observed are very minute, containing only a very few fibres of medium size. They run through the intermuscular septum, then dorsally under the skin to the organ. The first free organ of the trunk line is like the canal organs, large and flat topped. The organs diminish in size caudad and assume more nearly the characters of the smaller terminal buds, the free sensory surface being protected by the upward growth of the edges so as to give the whole organ a globose form with a narrow and shallow pore at the apex. The size of the fibres of the r. lateralis also diminishes as we pass caudad. Near the ganglion both proximally and distally of it, very large fibres predominate, some being as large as 12 micra in diameter, and the average is about 4. The very large fibres soon disappear and as far back as the level of the fifth spinal nerve the average size has diminished to six micra or less. The size of the fibres both here and in the case of the lateralis branches of the

VII nerve, seems to be correlated with the size of the organs to be innervated.

These branches of the lateral line nerve were all carefully traced and verified on the opposite side of the specimen plotted. They agree in detail with the description given above, except for slight variations in the arrangement of the anastomosing fibres. The cephalic and caudal portions of the second ramulus have separate origins from the r. lateralis and the caudal one only effects the anastomosis with the first spinal. There seems to be a subcutaneous anastomosis between the caudad portion and the third ramulus. A less thorough examination of other specimens shows that the general arrangement is the same. There is considerable variation in the details of the anastomoses, but there is always a very rich subcutaneous plexus in this whole region, arising from terminal branches of the r. lateralis, and the medial and dorsal rami of the spinal nerves and converging in the r. lateralis accessorius near the median line between the general dorsal musculature and the inter-spinal muscles.

Cole is certainly in error when he says ('98a, p. 169) that "it is certain that the true lateral nerve never anastomoses with the spinal nerves," if he uses the term anastomosis in the ordinary sense. There are, unqestionably, cases where branches of the lateralis vagi interdigitate with and for a short distance are bound up in the same sheath with branches of the spinals. But this temporary mingling of these dissimilar fibres has neither physiological nor morphological significance, and this is doubtless what Cole means by his statement.

Some further points bearing on the morphology of the ramus lateralis vagi are given in Section 12 under the head of the r. lateralis accessorius.

XI.—Summary of the Vagus Group.

These nerves, including the r. lateralis, receive visceromotor, communis, general cutaneous and acustico-lateralis fibres.

The viscero-motor fibres are of two types, coarse fibres from the nucleus ambiguus for the striated branchial musculature and fine fibres of unknown origin for the musculature (mainly unstriated) of the viscera. The communis fibres all terminate in the lobus vagi. They have three types of peripheral endings, viz.: (1) undifferentiated termini in the general visceral and mucous surfaces and more highly specialized termini in (2) taste buds and (3) terminal buds on the outer skin. The two latter classes of fibres are more numerous in the more cephalic branches of the complex, the former class in the more caudal branches, some of which they compose exclusively (intestinal and œsophageal rami). The general cutaneous fibres terminate in the spinal V tract and are distributed only to the outer skin by means of undifferentiated endings. The acustico-lateral fibres terminate mainly, if not wholly, in the tuberculum acusticum. They enter the brain only through the lateralis root and distribute only to organs of the lateral line.

The general scheme for the branchial nerves in Menidia may be expressed as follows: Each *truncus branchialis* arises from a special lobe of the general vagus ganglion and gives off the following rami.

1.—Pharyngeal rami, motor and sensory, supplying the pharyngeal muscles, taste buds, pharyngeal teeth, glands and mucous surfaces in general. They may arise from the truncus branchialis or from either the pre- or post-trematic ramus, or both, or from the ganglion directly. There is no branchiomeric uniformity as regards either their number or size, though the main sensory pharyngeal ramus is evidently typically associated with the pre-trematic ramus. These sensory fibres increase in number and importance as we pass caudad. They are reduced in the IX nerve.

2.—The pre-trematic ramus. Always very slender and absent in the case of the IX nerve. Probably wholly sensory.

3.—The post-trematic ramus. Divides before entering its gill into two ramuli, the dorsal purely sensory, the ventral mixed. The last post-trematic ramus, however, is single and sensory.

The internal and external pharyngo-clavicularis muscles are unquestionably innervated from the vagus and not from the first spinals as described for selachians and siluroids.

There are three rami cutanei dorsales of the vagus and these are all distinct from the r. supra-temporalis of the lateral line nerve, and from the associated communis fibres (vagal root of r. lat. acc.). They supply the skin of the dorsal part of the operculum and the parts adjacent. The most anterior (cephalic) of these rami is the ramus opercularis vagi and it anastomoses with the r. opercularis superficialis facialis.

A true trapezius muscle is present in Menidia, being innervated from the vagus and not from the first spinal, as other authors have described for other species of teleosts. Its nerve may be homologized with the n. accessorius Willisii.

The ramus lateralis vagi receives lateralis fibres from the tuberculum acusticum and communis fibres from the IX root. The latter correspond to the vagal root of the r. lateralis accessorius of some other fishes. Its first branch, the r. supra-temporalis, receives both components. It distributes its lateralis fibres to two canal organs, the one in the supra-temporal commissure and the other in the main canal between that commissure and the opercular canal. Part of the fibres supply one or two naked sense organs which lie just dorsally of the main

canal. The communis fibres join, in two branches, the r. lateralis accessorius. The succeeding ramuli of the ramus lateralis supply each one organ of the lateral line, beginning with the first one behind the occipital commissure. The first and second ramuli each contain communis fibres also, which communicate with the r. lateralis accessorius. The naked cutaneous sense organs not in the lateral lines supplied by these nerves I have regarded as terminal buds and they are so drawn in the figures; but subsequent study of Gadus rather favors the idea that they are pit-organs of the lateral line series.

Section 6.—The Auditory Nerve.

The ear of Menidia is highly developed. The auditory nerve is, accordingly, large (Fig. 5). There is an incomplete division at its origin into caudal and cephalic rami, the former being intimately associated with the n. lateralis vagi, the latter with the lateralis roots of the VII nerve. The auditory root fibres are so intricately mingled with those of the roots just mentioned that their intracranial courses could not be analyzed.

There is a considerable bundle of coarse fibres passing from the dorsal edge of the ramulus acusticus ampullæ anterioris near its separation from the rest of the cephalic auditory root dorsally to joint the dorsal lateralis root of the facialis. It runs externally to all of the other roots of the V + VII + VIII complex and crowded in closely between the communis root of the facial and the recessus utriculi (Fig. 20, *VIII—d. l.*) In one of my series I found what appears to be a similar twig from the VIII running along the inner side of the ganglionic complex to the ventral lateralis root of the facial (shown in Fig. 20 just dorsally of the motor root). But as this could not be verified

in other series of sections it remains an uncomfirmed observation. Allis ('97, p. 624) describes an anastomosis between the r. ampullæ posterioris of Amia and the lateralis component of the IX root. As in the present case, the farther course of the fibres could not be followed.

The apparent origin of all of the acustico-lateral fibres from the tuberculum acusticum is a single fibre complex, the separation into the roots as we enumerate them taking place just before their emergence from the oblongata. The auditory rami break up to supply the papilla acustica lagenæ, the three cristæ acustici, the macula neglecta, the macula acustica sacculi, and the macula acustica recessus utriculi. The glossopharyngeal nerve runs along the inner face of the caudal ramus and as it emerges from the cranium passes between the ramulus ampullæ posterioris and the ramulus lagenæ just at their point of separation. There is no exchange of fibres between the VIII and the IX nerves. The r. lateralis vagi arises at the same transverse level as the caudal acoustic ramus but farther dorsal. It is crossed externally by the ramulus ampullæ posterioris but, though the two nerves are in contact for a considerable distance, there is no anastomosis. The cephalic ramus arises at the same transverse level as the VII nerve and its ramuli closely follow the outer face of the V + VII ganglionic complex. In this case also I think that there is no interchange of fibres.

Ganglion cells are not found in the auditory roots until they begin to break up into their ramuli, beyond which point they are freely scattered among the fibres reaching in some cases quite to the sensory epithelium. The cells are exceedingly minute, smaller than those of the lateral line ganglia and scarcely larger than the diameter of the medullary sheaths. The calibre of the

fibres of the auditory root varies widely, some fibres being very coarse, as large as those of the lateral line roots, though most are of medium size, while some are quite fine. The latter are scattered among the coarser ones in rather close bundles at first. Inasmuch as it was not possible to trace the different kinds of fibres to distinct origins centrally, nor to follow them with precision through their ganglia, the detailed account of the ramuli will hardly be necessary, for the descriptions of Retzius ('81) amply cover the topographical relations. These relations are shown on Figs. 5, 17, 18, 19, 20, 21.

Section 7.—The Trigemino-Facial Complex.

The fifth and seventh nerves have been confused in the descriptions of the earlier writers upon the bony fishes and such confusion was inevitable so long as the method of dissection alone was relied upon. Those who have studied these nerves microscopically have confined their observations to their root portions, and because of their ignorance of the peripheral courses of these roots have often been led into errors of interpretation. I have succeeded in following all of the facial and trigeminal roots through the ganglionic complex with precision and most of the fibres have been traced to their peripheral endings with equal certainty. The roots of the fifth and seventh nerves are as distinct in Menidia as in any vertebrate, all of the fifth roots emerging at one transverse level and the seventh at another. These two root complexes are, however, so close together and so intimately joined immediately after their exit from the brain that their exact analysis would be impossible by dissection. All of their ganglia fuse into one mass and are indistinguishable

macroscopically, the entire complex being termed by many writers the Gasserian ganglion. The microscope, however, readily analyses this complex and distributes the fibres from each element into their respective rami (Figs. 4, 11 and 12).

The analysis of the rami peripherally is a much more difficult matter. Nevertheless it has been accomplished with results which I think are free from ambiguity except in a very few points. There are very few of the rami which can be assigned to either the fifth or the seventh nerve alone. In a few cases, such as the r. palatinus VII, there is but one component represented; but in the majority of cases fibres from both the fifth and seventh roots are bound up in the same nerve trunk and the question of nomenclature becomes a perplexing one. The time will undoubtedly come, as Strong has pointed out, when the nomenclature of the cranial nerves must receive a thorough revision. The time is certainly not ripe for this now, and I have avoided, so far as possible, the introduction of new names, selecting from the current terms the one which seems the most appropriate in each case, and giving to it a definite and often somewhat arbitrary significance. Thus I have assigned, following the usual custom, the truncus hyomandibularis to the seventh nerve, though it receives general cutaneous fibres from the trigeminus root in addition to its proper facial fibres. Similarly the r. maxillaris trigemini receives communis fibres from the facial root.

As just intimated, the composition of the several rami at their proximal ends can be stated with accuracy. Peripherally the three classes of sensory fibres and the motor fibres can, with few exceptions, again be easily separated before they pass to their terminal organs. In the nerve

trunks the coarse fibres and the fine fibres can be separately followed, but it is often not possible to keep the two classes of fine fibres (communis and general cutaneous) distinct throughout their courses, nor always the two classes of coarse fibres (lateralis and motor). Nevertheless their terminal relations enable us to infer their courses in the trunks with sufficient accuracy.

I.—The Roots and Ganglia of the Facialis.

The facial has four roots emerging at the same transverse level, a ventral motor root, widely separated from the others, ventral and dorsal lateralis roots and the communis root, emerging between the two last. The last three issue high up towards the dorsal side of the oblongata.

1.—The Motor Root.

The motor VII nucleus lies near the median line in contact with its fellow of the opposite side and separated from the fourth ventricle by fibres of the fasciculus longitudinalis dorsalis only. The cells are large and are grouped on each side in two clusters, from each of which a strong tract of fibres (secondary tracts or dendrites ?) runs out laterally and ventrally into the lateral white columns. In connection with the more mesal one of these tracts are fibres which seem to run from the fasciculus longitudinalis dorsalis to the VI root. The motor VII nucleus occupies the position of the commissura accessoria of Mauthner, which is interrupted in this region.

From both parts of this nucleus facialis fibres run dorsad and form a large, close bundle on the lateral aspect of the fasciculus longitudinalis dorsalis. It lies just ventrally of the bundle of fibres derived from the nucleus ambiguus,

as described above, and soon fuses with it. Like the latter, these fibres are of finer calibre than those of the fasciculus, which are very large. The fibres of the VII nerve soon withdraw from the fasciculus, but pass forward parallel with it for some distance. Whether the facial root receives additions from the fasciculus or contributes to it could not be determined. Presumably there is some relation by collaterals or otherwise, as the union of the fibres of the root and the fasciculus is here very intimate.

This arrangement of the motor VII root is substantially identical with that described by Goronowitsch in Acipenser ('88, p. 498). Johnston ('98) finds by the Golgi method in Acipenser that part of the motor VII root arises directly from its nucleus and a larger part from the fasciculus longitudinalis dorsalis. The entire course of these latter fibres apparently, was not impregnated, so that it remains uncertain whether they arise from cells of the VII nucleus farther back or whether they belong to the fasciculus proper.

At 577 (Fig. 19) the facialis fibres turn abruptly toward their exit. This motor root immediately after its exit from the oblongata joins the ventral lateralis root proximally of its ganglion and follows the ventral surface of that root, as shown on the plots.

2.—*The Communis Root.*

The communis root of the facialis passes into the fasciculus communis and constitutes the whole of the preauditory portion of that tract (Fig. 4, *f. c.*). It emerges from the oblongata closely wedged in between the dorsal and ventral lateralis roots (Figs. 19, 20, *com. VII*, Fig. 11) and passes, still between those roots, ventrally into the geniculate ganglion. This ganglion is of an elongated

pear shape with the larger end down. It is bounded above by the dorsal lateralis root and ganglion, in front by the Gasserian ganglion, below and behind by the ventral lateralis root and ganglion. The cells of the geniculate ganglion vary greatly in size. The largest ones are fully 50 micra in diameter, while the smaller ones are scarcely more than 10. The large and small cells are irregularly mingled with a tendency for the small cells, which are less numerous than the large ones, to gather in the interior of the ganglion (Fig. 21, *gen. g. VII*).

3.—The Lateralis Roots.

The two lateralis roots of the facial immediately upon entering the oblongata fuse and enter the tuberculum acusticum together, the fibres of the dorsal root farther cephalad than those of the ventral one. These root fibres throughout their internal course are so intimately mingled with the most cephalic fibres of the VIII nerve that analysis is impossible.

After emergence from the oblongata the two lateralis roots at once diverge and remain distinct throughout their entire peripheral courses. The fibres from the dorsal lateralis ganglion pass into the r. ophthalmicus superficialis facialis and the r. buccalis; those from the ventral ganglion all pass into the truncus hyomandibularis. The cells of these ganglia are very small, about 20 micra in diameter, and as a rule only two or three times the diameter of their fibres. They are usually not crowded, but, like those of the lateral ganglion of the vagus, they are scattered among the fibres (Figs. 4, 5, 11 and 19–22). The dorsal root, before reaching its ganglion, receives the small bundle of fibres from the VIII root, as described in Section 6 and figured in Fig. 20.

II.—The Roots and Ganglion of the Trigeminus.

The sensory and motor roots of the trigeminus issue high up on the side of the oblongata and so intimately fused that their separation is not easy, even microscopically (Figs. 4, 12, 20–23).

1.—The Motor Root.

The motor nucleus of the trigeminus lies in the floor of the ventricle laterally of the fasciculus longitudinalis dorsalis and somewhat removed from it. Both the motor nucleus and root lie farther dorsally than the spinal V tract and at their exit the motor fibres are dorsal, a relation, however, which is not maintained peripherally. A broad band of fibres arising apparently in the motor nucleus of the opposite side passes through the nucleus and doubtless contributes to the root. It also passes through the fasciculus longitudinalis dorsalis and may derive some fibres from that source.

The motor root passes through and mingles with the general cutaneous root so intimately that it is difficult for a short distance from their exit to distinguish them. Doubtless fibres from other than the motor nucleus, the sensory nucleus and the spinal V tract enter the V nerve, but as they cannot be separately followed peripherally, I have devoted no especial attention to them. Though the general cutaneous and motor fibres cannot be clearly separated for a short distance after their exits, they can be separated both centrally and peripherally of this point, so that their entire courses can be given with all needful accuracy. In the case of none of the other components which participate in the V + VII ganglionic complex is there any difficulty in distinguishing them and following them throughout the complex.

2.—*The General Cutaneous Root.*

Immediately upon entering the brain the sensory root of the trigeminus divides. One part joins the motor V to form the deep root (*d. V*, Fig. 20) and goes to the chief sensory trigeminal nucleus. The larger part turns caudad and forms the whole of the pre-vagal spinal V tract. Its internal course has been described in Section 3. It crosses the motor trigeminus root and forms the most dorsal member of the V + VII root complex. It is then crossed externally by the dorsal lateralis root and ganglion. The Gasserian ganglion lies farther cephalad than any of the other members of this complex. Its cells resemble those of the geniculate ganglion, though the largest ones are rather larger.

III.—Comparative Review of the Trigemino-Facial Roots.

Stannius enumerates forms with five and forms with four roots in the trigemino-facial complex and considers the latter as the more characteristic of fishes, counting the two lateralis roots as one. His analysis of these roots is remarkably exact, more so than those of some more recent writers who have had better methods at their disposal. Comparing Menidia with his forms with five roots, it appears that his first root is our general cutaneous plus motor V, *i. e.*, as he states, the trigeminus in the strict sense; his second root is our dorsal lateralis root; his third root, our ventral lateralis root; his fourth root, the communis root; and his fifth root is our motor VII.

Goronowitsch's endeavor in both the earlier ('88) and the later ('96) papers to elaborate a simple serial arrangement of dorsal and ventral roots of which the several pairs should be strictly homodynamous with each other and with spinal roots must be reckoned a total failure, as

he has not taken into account the qualitative differences in the fibres of these roots. The second paper is dominated by this attempt quite as much as the earlier one and Strong's suggestion ('95, p. 168) that the ventral root of Trigeminus II is not motor but sensory (lateralis) is rejected. Nevertheless the papers of Kingsbury ('97) and Johnston ('98) both show that in Acipenser the composition of these nerves is just as in Menidia, viz., Trig. I is the general cutaneous and motor V roots, Trig. II is the two lateral line roots of the facial nerve, and the facial of Goronowitsch is the communis and motor VII roots. See sub-section XII beyond.

IV.—The Truncus Hyomandibularis.

This nerve receives all of the motor VII root, all of the fibres from the ventral lateralis ganglion, a bundle of communis fibres from the geniculate ganglion, and a bundle of general cutaneous fibres from the Gasserian ganglion. Of these the lateralis component is by far the largest, the motor bundle is about one-fifth as large, while the other two components are about as large as the motor. The motor fibres join the lateralis root from the ventral side snd mingle with them. Beyond the lateralis ganglion several small bundles of fine communis fibres are derived from the geniculate ganglion (not shown on any of the plots). Still farther cephalad and just as the truncus is passing through its foramen it is crossed by the large r. palatinus and from the latter receives a large bundle of communis fibres. These constitute a compact bundle on the cephalic face of the truncus and as such can be followed almost the entire length of this nerve, constituting peripherally the r. mandibularis internus VII.

As the truncus hyomandibularis is passing through its

foramen, it crosses a ganglion of the sympathetic chain (Fig. 21, *sy.*) and may derive some fibres from it. After emergence from its foramen it runs outward to the hyomandibular bone and then turns abrubtly ventrad and slightly caudad, entering a canal in that bone.

At this point, *i. e.*, just as the truncus is turning ventrally and some distance outside of the foramen, it receives on its caudal face a considerable bundle of fine fibres which come from the Gasserian ganglion. See Fig. 4. Their course is as follows: The truncus hyomandibularis and the r. palatinus together issue from a foramen which is distinct from that of the V nerve and the remainder of the VII. But a small portion of the Gasserian ganglion extends far caudad nearly to this foramen. Here there is a separate foramen in the cranial wall just cephalad of that of the truncus hyomandibularis through which there passes a large blood vessel and also a narrow tongue of ganglion cells belonging to this caudal tip of the Gasserian ganglion (Fig. 2, *G*). From this little extra-cranial ganglion a considerable bundle of fibres passes out laterally (510) until it reaches the hyomandibular bone (Figs. 2, 4, *t. f. 2*). It then turns caudad and ventrad and is soon joined by another similar bundle of fine fibres (Figs. 2, 4, *t. f. 1*) which is derived from the extreme cephalic end of the Gasserian and which emerges from the cranium with the great infra-orbital trunk, curving back and pursuing a tortuous and rather peculiar course, which will be more fully described in connection with the infra-orbital trunk.

The combined general cutaneous component now passes back and joins the truncus hyomandibularis on its caudal face after it has entered the canal in the hyomandibular bone and begun to turn ventrad (530). Beyond this point it can be separately followed in the truncus with great

ease, for its fibres do not mingle with the other fine fibres, but are separated by the coarse lateralis and motor fibres from the communis bundle.

1.—*The Ramus Opercularis Profundus VII.*

The motor component of the truncus hyomandibularis can be followed, in spite of its intimate relations with the lateralis fibres, up to the point where the truncus hyomandibularis begins to turn ventrad (515). Here it divides into three parts, one part leaving the main nerve dorsally to turn immediately caudad, another continuing cephalad after the truncus has turned ventrad, while the third follows the truncus in its farther course. The first two parts I include under the term r. opercularis profundus VII.

The dorsal branch, which is the r. opercularis of Stannius, supplies three muscles. It passes directly caudad just dorsally of the apex of the narrow slit-like extension of the pharyngeal cavity which runs up between the pseudobranch and the first gill. It then passes through the dorsal end of the m. adductor hyomandibularis from its mesal to its lateral face, meantime contributing a few fibres for the innervation of this muscle (*m. ad. hy.*, Fig. 4). The nerve continues caudad along the outer face of this muscle to its end, and then farther caudad between the m. adductor operculi and the m. levator operculi, where it divides, the ventral twig supplying the m. adductor operculi (*m. ad. op.*). The remaining fibres continue caudad and almost immediately cross the course of the r. opercularis vagi. The two nerves lie almost in contact for a short distance, but clearly do not anastomose, as is the case in the carp (Baudelot, '83, p. 132, and Stannius, '49, p. 61) and Lophius (Guitel, '91). A little farther

caudad these remaining fibres from the r. opercularis VII enter and innervate the m. levator operculi (*m. lev. op.*).

The motor fibres which continue cephalad from the truncus hyomandibularis soon turn ventrally to reach the dorsal surface of the very large m. adductor arcus palatini (*m. ad. a. p.*), in which position they continue cephalad, giving off twigs for the innervation of this muscle from time to time. This ramus for the adductor arcus palatini is larger than the entire dorsal portion of the r. opercularis profundus.

2.—The Ramus Opercularis Superficialis VII.

Immediately after the truncus hyomandibularis has entered the canal in the hyomandibular bone it gives off a ramus directed caudad, the r. opercularis superficialis VII (*op. s. VII*), which at once separates into two, a dorsal and a ventral. The two portions pass through a common foramen in the opercular bone. The ventral one contains only coarse lateralis fibres and supplies the penultimate (6th) canal organ of the opercular canal (*op. s. VII. 1*). In some cases it has a separate origin from the truncus.

The dorsal portion contains coarse and fine fibres. The former, comprising about four-fifths of the area of the cross-section of the nerve, are lateralis fibres; the latter are derived almost exclusively from the general cutaneous component before it has yet fused with the truncus. Upon reaching the preopercular bone about three-fourths of the coarse fibres are drawn off (*op. s. VII. 2*), enter a foramen in that bone and supply the last (7th) canal organ of the opercular canal. The remaining coarse fibres and all of the fine ones continue caudad as a compact round bundle between the preopercular bone and the ventral tip of the hyomandibular.

In this position it begins to give off small branchlets of a few fibres each. One of these (*op. s. VII. 3*) of fine fibres leaves at 580 and turns dorsad, curving around the caudal projection of the hyomandibular bone where it articulates with the opercular, and continues caudad along the dorsal and inner surfaces of the latter bone. Here it joins (600) the fourth branch of the r. opercularis vagi, as described under that nerve, and the combined nerve supplies the adjacent mucous lining of the operculum.

Another branch of the r. opercularis superficialis VII is somewhat larger than the last and is composed of coarse fibres (*op. s. VII. 4*). It originates at the same point as the last mentioned and passes caudad and dorsad by a circuitous path around the base of a scale to a naked sensory papilla (*o.5*) situated a short distance caudad of the seventh opercular canal organ and in a line continuing the direction of the sixth opercular pore.

The fifth branch (coarse and fine fibres, *op. s. VII. 5*) leaves also at the same point. It immediately divides into several branches and supplies the skin of the operculum laterad and ventrad and caudad of the point of origin.

The ramus opercularis continues ventrad and caudad between the opercular bone and the skin and the sixth branch, containing coarse and fine fibres, separates dorsally at 612. This branch can be traced nearly to the caudal edge of the operculum, running dorsally of the main nerve and nearly parallel with it. The finer fibres are given off to the skin early in its course. The termini of the coarser ones, as in the last case, could not be determined by reason of defects in the sections.

The main nerve proceeds to the extreme ventro-caudal edge of the operculum, giving off several small branchlets

similar to the last, and finally terminates in two large naked sensory papillæ (*0.3* and *0.4*). Most of the fine fibres are given off with the earlier branches, but some seem to persist quite to the end of the nerve. The coarse fibres, including those which supply the three sense organs described, as well as those which go out with the other ramuli and were not traced to sense organs, are somewhat smaller than the largest lateral line fibres, but much larger than any communis fibres.

The operculum is covered with large scales; it is, therefore, difficult to get perfectly continuous series of sections of the skin and my sections are imperfect in the middle part of the operculum, so that, while the trunk of this nerve can be followed easily, yet its fine cutaneous branchlets are usually lost before they reach their final distribution.

On the opposite side of the specimen plotted branches of the r. opercularis superficialis VII were traced to four naked cutaneous sense organs. One corresponds to the organ *0.5* of the plots, and another is somewhat behind this and a little ventrally of it. Two organs were found near the ventral edge of the operculum and lying somewhat cephalad of those figured on the plots. The second may correspond to the first of the plots, or both may lie in front of the latter.

Having been led to believe from the appearance of my sections that more perfect preparations would reveal a larger number of cutaneous sense organs on the operculum, I made surface preparations of the operculum of a number of specimens. The operculum of a small specimen which has been preserved in 10 per cent formalin can easily be removed and examined as a transparent object, particularly if the pigment-bearing mucous lining of the inner surface be first stripped off. Fig. 27 is a camera sketch of such a preparation examined in water without staining or clearing and controlled by several similar preparations, as well as others stained in various ways.

The r. opercularis superficialis VII and its principal branches can easily be followed, and it is seen to supply four naked sense organs along the lower margin of the operculum (*o.1* to *o.4*). They are large and flat and lie upon or below the lowest row of opercular scales in a line which is the direct continuation caudad of the horizontal limb of the opercular canal, this line passing through the fourth opercular pore. The number found was uniformly four, except in one case, where the first one seemed to be double. The arrangement varies somewhat in different specimens, but never deviates greatly from that figured. The two organs found in the sections and figured on Fig. 3 are probably numbers 3 and 4 of the series. The naked organs along the outer surface of the opercular canal, to be described presently, could also be distinctly seen. Their number and arrangement vary greatly in different specimens, but they are always smaller than the four organs supplied by the r. opercularis and when examined in the unstained specimens with a low power look like little discs with a brilliant highly refracting centre.

The organ lying behind the sixth opercular pore was seen in a few instances. It may be followed by other organs behind, as suggested above, for the preparations here are opaque and confused by reason of the thick origin of the underlying m. dilator operculi.

Regarding the morphology of these five or six naked opercular organs, I think there is but one conclusion possible. That they belong to the lateral line rather than to the communis system is shown by their innervation. See further, Section 2, III.

Stannius makes no mention whatever of the r. opercularis superficialis. That it is not contained in his r. opercularis VII is shown by the fact that he correctly regards that nerve as exclusively motor; *i. e.*, his r. opercularis is our r. opercularis profundus only.

After giving off the r. opercularis superficialis VII, the truncus hyomandibularis continues directly ventrad in a canal of the hyomandibular bone until the ventral edge of

the bone is reached. Here it divides into the r. hyoideus and the larger r. mandibularis. The latter turns abruptly cephalad, leaving the canal and running along the outer surface of the bone to its cephalic tip.

3.—The Ramus Hyoideus VII.

The ramus hyoideus (*hy.*) runs ventrad and caudad. Its first branch (*hy. 1*) separates at once and might be considered a separate nerve. It contains only coarse fibres, enters the lateral line canal of the preoperculum and supplies the fifth opercular canal organ.

The remaining and larger portion of the r. hyoideus contains coarse and fine fibres and descends along the inner face of the preopercular bone, and lower down along the inner side of the interopercular bone, nearly to the first (most dorsal) branchiostegal ray. Here it divides into two approximately equal divisions, each with both coarse and fine fibres (560). Of these one, which may be regarded as the main ramus, turns slightly cephalad, the other (*hy. 2*) continues caudad and ventrad and will first be described. It follows dorsally a big blood vessel running along the inner face of the interopercular bone, giving off at once a small twig which later rejoins the main trunk, then several smaller branchlets which could not be traced, and at 610 a slightly larger branch of fine fibres with a few coarse ones, which passes directly dorsally between the subopercular and interopercular bones to emerge upon the skin dorsad of the latter bone. It distributes to the skin covering the cephalic half of the suboperculum. The remainder of this nerve breaks up in the branchiostegal membrane, which it innervates.

The cephalic or main division of the r. hyoideus passes ventrad in two branches, each with coarse and fine fibres,

one large one mesally of a big vessel and a minute twig laterally of it. The latter (*hy. 3*) runs down along the inner face of the interopercular bone and at about the middle of that bone divides. One ramulus pierces the bone and supplies the overlying skin cephalad of this point; the other ramulus continues forward in the original position along the inner surface of the interopercular and finally divides again, both parts piercing this bone to end in the skin covering it. It can be traced forward under the eye up to 400. The ramuli of this more lateral twig supply practically the whole cutaneous area over the interopercular bone.

The more mesal branch of the r. hyoideus runs forward along the dorsal limit of the branchiostegal membrane under the bones of the hyoid arch. Here it breaks up to supply the branchiostegal muscles (*hy. 4*, m. hyoideus of Vetter) and the surfaces of the branchiostegal membrane. One twig of the latter type (*hy. 5*) can be followed nearly to the cephalic end of the ceratohyal bone (350).

The coarse fibres of the r. hyoideus are mainly distributed to the branchiostegal muscles, but some of them go out to the skin with the general cutaneous branches. Stannius mentions ('49, p. 62) that movement of the branchiostegal membrane follows stimulation of the truncus hyomandibularis. No cutaneous sense organs were found in the areas supplied by any of the branches of the r. hyoideus. The fine fibres of this nerve are derived exclusively from the general cutaneous nerve bundle, *i. e.*, the r. communicans from the Gasserian ganglion. They comprise, moreover, the whole of that component except the portion already given off to the r. opercularis superficialis. This can be demonstrated with ease, for the general cutaneous component of the truncus hyomandibu-

laris runs down its caudal and ectal side, while the communis component runs down the cephalic and ental face of the truncus, the two fine-fibred portions being separated through the whole length of the truncus by the coarser lateralis and motor fibres. It is only at the extreme ventral end of the truncus that there is any possibility of any considerable intermingling of their fibres. Here, though the two fine-fibred components lie rather close together, yet the sections show plainly that they do not exchange fibres. We have, therefore, good anatomical evidence that no considerable number of communis fibres enter the r. hyoideus, and that independently of the fact that no terminal buds are found in its course. It is equally clear that no considerable number of general cutaneous fibres enter the r. mandibularis VII, and this is substantiated by the fact, to be noted below, that the areas in which the special cutaneous fibres of the r. mandibularis VII find their terminal organs receive an independent general cutaneous nerve supply from the r. mandibularis V (see Fig. 3).

4.—The Ramus Mandibularis VII.

This nerve (*man. VII*) contains lateralis and communis fibres, which though bound up in the same trunk for most of their courses, nevertheless correspond to the r. externus and r. internus of the Amphibia and of some other fishes.

After its separation from the r. hyoideus and immediately upon its emergence from its canal in the hyomandibular bone it sends a very small branch (*m. VII. 1*) around the upper edge of the preoperculum, then ventrally between this bone and the m. adductor mandibulæ. Here it divides, the smaller part (of coarse fibres) running laterally through this muscle to the skin, close under

which it turns dorsad around the base of a scale to a naked sense organ (*o.6*) lying on the outer face of the scale and just overlapped by the free edge of the next dorsal scale. The larger part of this first ramulus continues ventrally along the outer face of the preopercular bone to the opercular canal. It enters the bony canal and just dorsally of the membraneous canal it divides, a minute twig turning cephalad, the larger portion caudad. The latter supplies four naked organs on the outer skin covering the most caudal part of the horizontal limb of the opercular canal and the base of the fourth pore of that canal. The former supplies a single similar but larger organ on the base of the third pore. The fibres of this ventral part stain very intensely, like lateralis fibres, though they are of small size.

On the opposite side of the specimen figured this first branch pursues a course similar to that just described except that all of its fibres pass through from the mesal to the lateral face of the m. adductor mandibulæ before it divides into its dorsal and ventral ramuli. The ventral ramulus then passes down the outer face of the muscle instead of its inner face to reach the opercular canal. Here it supplies four naked organs with its caudal twig, but the cephalic twig, after supplying a naked organ just caudad of the third opercular pore, continues cephalad, receives a considerable addition from the second branch of the r. mandibularis VII just before the latter enters the fourth opercular canal organ and then supplies a large sense organ lying just cephalad of the third opercular pore.

All of the naked organs supplied by this branch, as well as similar ones farther cephalad, to be described immediately, are of the same nature as the similar but larger organs supplied by the r. opercularis superficialis and I homologize them with the pit-lines of ganoids. They correspond, doubtless, to the similar lines mentioned by

Allis ('97, p. 632) in Gadus and Esox, and all of these may represent the gular line of Chlamydoselachus (Garman, '88).

The r. mandibularis VII after giving off its first branch, as just described, runs along the outer face of the hyomandibular bone. In this part of its course it contains chiefly very coarse lateralis fibres, with a small bundle of fine communis fibres along its inner face. At the extreme cephalic tip of the hyomandibular bone (500) the fine fibres separate mesally and dorsally from the coarse ones and a strong fascia in which portions of the m. adductor mandibulæ are inserted, passes between the two divisions, which become widely separated. The fibres of the more lateral bundle are very large with few medium ones and a very few small ones intermingled; those of the mesal bundle are mostly very small with a considerable number of medium-sized and more heavily myelinated fibres intermingled. These bundles represent the r. mandibularis externus and the r. mandibularis internus of the facial nerve.

From the lateral bundle the second branch is given off at 488 (*m. VII. 2*), which descends along the inner face of the preopercular bone, running slightly caudad, to enter the opercular canal and finally to supply its fourth canal organ.

The third branch arises from the fine-fibred communis bundle at 483 (*m. VII. 3*), runs ventrad and caudad and somewhat further mesad than the last. Having reached the mucous lining of the pharyngeal cavity just dorsally of the epihyal bone, it divides into two branches, directed cephalad and caudad respectively. The former runs under and innervates the mucous membrane covering the dorsal surface of the bones of the hyoid arch as

far forward as the cephalic end of that arch (400). The latter distributes in the same way to the mucosa overlying the hyoid bones as far back as their articulation with the hyomandibular. The epithelium in the region supplied by this nerve is thicker than the adjacent pharyngeal lining and is thrown into deep folds, giving the appearance characteristic of a sensory surface, but no taste buds, such as are abundant on the dorsal surfaces of the gill arches at the same levels, were found, though careful search was made for them in several specimens along the whole length of the hyoid arch. This region is, however, more richly supplied with gland cells than usual and these fibres not improbably are distributed in part to them. It is important to note that the corresponding region in Amia is innervated from the fourth branch of the r. mandibularis V (Allis, '97, p. 612).

The two bundles of the r. mandibularis VII continue forward between the m. adductor mandibulæ and the symplectic bone, just above the dorsal edge of the preopercular bone. In this position they again unite (465) and there is more or less confusion of the fibres of the two bundles. After their union they give off the fourth branch (*m. VII. 4*), which is composed chiefly of medium-sized fibres and a few very large ones. It arises apparently wholly from the lateralis component and takes its course ventrad and slightly caudad along the outer face of the preoperculum to the opercular canal. It pierces the bony canal, the fibres running dorsally and laterally of the membraneous canal to supply three naked organs lying in the skin covering the canal, two caudad of and one cephalad of the third opercular canal organ.

On the opposite side of this specimen the fourth branch and the corresponding sense organs caudad of the second

opercular pore are totally wanting, though it should be noted that on that side the first and the sixth branches supply each an additional naked sense organ and that the latter encroaches somewhat upon the area supplied by the fourth branch.

A short distance farther cephalad the fifth branch (*m. VII. 5*) of the r. mandibularis VII, composed wholly of coarse fibres, separates, takes a course nearly parallel to that of the fourth branch and enters the opercular canal to supply its third organ.

At almost the same point as the last the sixth branch (*m. VII. 6*) arises. It is composed of medium or small fibres with very densely staining sheaths of the same type as those of the first and fourth branches. It passes directly ventrad and into the bony opercular canal by a separate foramen in the preopercular bone. It then turns cephalad, running dorsally of the membraneous canal, and supplies three naked sense organs, two lying superficially of and a short distance caudad of the second and first organs of the opercular canal respectively, and one just cephalad of the latter. The origin and course of this branch are strictly analogous with those of the first and fourth branches, which it closely resembles.

On the opposite side this branch supplies four naked sense organs, two cephalad of the second opercular pore and caudad of the second opercular canal organ, one cephalad of the first opercular pore and immediately caudad of the first canal organ, and one cephalad of the first canal organ.

In another specimen we have an arrangement of the first six branches of the r. mandibularis VII which differs from either side of the specimen figured. The two sides of this specimen agree in the following arrangement. The first branch is represented only by its dorsal twig for

the organ *o.6*. The fibres for the other naked organs supplied by this branch in the specimen figured are supplied by a branch arising cephalad of the second branch (the third branch of this specimen). Branches 1 and 2 arise apparently from the coarse-fibred component. Between the second and third branches in this specimen the nerve splits into the fine-fibred and coarse-fibred bundles as in the other case, and the third branch arises from the coarse-fibred bundle. The fourth branch is absent. The fifth arises from the coarse-fibred bundle just as it is rejoined by the fine fibres. The sixth branch supplies two organs caudad of the second canal organ and doubtless some cephalad. Its ultimate course was not traced.

At 430 the seventh and eighth branches of the r. mandibularis VII are given off together. They both arise apparently from the coarse-fibred component and pass ventrad to the dorsal surface of the bony opercular canal. The eighth branch turns cephalad along the dorsal surface of the preopercular bone, while the seventh, which is four or five times as large, passes through a foramen into the bony canal at once, thence cephalad to supply the second and first organs of the opercular canal.

The seventh branch is composed of very coarse fibres; the eighth branch chiefly of medium fibres, with some fine ones intermingled. The latter branch continues cephalad along the dorsal and outer surface of the preopercular bone and under the fleshy origin of the m. adductor mandibulæ. It increases in size cephalad and ultimately joins a branch of the r. mandibularis V (*c. V–VII. 1*). This branch very clearly contains fibres from both the facial and the trigeminus. The former continue forward after the anastomosis with another trigeminal branch and finally supply three naked sense organs along the mandibular canal. They will be further discussed in connec-

tion with the trigeminal branch with which they are distributed.

After giving off the last two branches the r. mandibularis VII continues forward along the outer surface of the symplectic and almost immediately divides into a ventral bundle composed exclusively of very coarse fibres and a smaller dorsal bundle of very fine fibres with a few of medium size. These bundles become quite widely separated, a few fibres of the m. adductor mandibulæ lying between them. The ventral bundle comes to lie in a deep groove in the symplectic toward the cephalic end of the latter, which finally becomes a closed canal, while the dorsal bundle enters a separate canal lying farther dorsally and bounded by the symplectic, the mesopterygoid and the quadrate. The dorsal bundle almost immediately emerges on the mesal side of the bones and continues cephalad along the inner face of the quadrate; but the ventral division runs much farther cephalad in its canal, a process of the symplectic containing the canal running forward along the inner face of the quadrate to receive it. It also finally emerges on the inner face of the quadrate and here the two bundles re-unite (290). From this point cephalad the two bundles run along the inner face of the quadrate in contact but without mingling of fibres and in this relation several very fine ramuli (not shown on the plots) are given off from the finer bundle to the mucous lining of the mouth immediately adjacent to the nerve trunk. No taste buds can be found in this region, nor is the epithelium so glandular as in the corresponding region overlying the hyoid bones and supplied by the third branch of this nerve.

Farther forward, along the inner side of the articular and dentary bones, the coarse and fine-fibred bundles are

more intimately united, though from time to time they may completely separate for a short distance, as far forward as the cephalic tip of the articular bone. Here the two divisions finally separate (165), the dorsal one, as before, containing fine and medium fibres, while the ventral one contains all of the very coarse fibres with a few of medium size.

Previously to this, however, a coarse-fibred twig has been given off (200) to supply the fifth canal organ of the mandibular canal (*man. VII. 9*).

The fine-fibred dorsal division (*man. VII. 10*) will first be described. It follows, parallel to the other division, the mesal surface of the mandible close under Meckel's cartilage, and at 120 gives a rather strong branch dorsad which supplies numerous taste buds of the mucous lining of the mandible. These fibres were definitely traced in several cases to the buds and not merely to regions where buds abound. This epithelium is also very glandular and doubtless many of the fibres are not destined for the sense organs.

Other similar branches go off cephalad, also branches for the floor of the mouth over the intermandibularis muscle, which is richly supplied with taste buds. The taste buds supplied by this nerve become more numerous as we pass cephalad, being very abundant all over the inner surface of the lower lip. These buds resemble in structure those farther back on the palate and gill arches supplied by the IX and X nerves, being flask-shaped, resting on a high dermal papilla or fold and with the neck of the flask projecting above the epithelium.

The origin of the intermandibularis muscle separates this dorsal division from the remainder of the mandibularis VII, which lies ventrally of this muscle. At the tip

of the mandible a branch of the dorsal division enters the alveolar canal of the dentary bone and apparently supplies its teeth.

Returning now to the coarse-fibred ventral division (165), it follows parallel with the dorsal division the inner face of the mandible and at about 150 gives off ventrally about one-fourth of its fibres, comprising some very coarse ones and some of the medium size (*man. VII. 11*). At 130 these fibres pass through a foramen in the dentary bone into the mandibular canal, where they supply the fourth mandibular canal organ.

At the level of that organ (120) the main nerve is joined by a branch of about the same size which is derived from the r. mandibularis V (*V–VII. 2*) and passes ventrad between the articular bone and Meckel's cartilage to the dorsal surface of the r. mandibularis VII. It is composed of fine, medium and very coarse fibres, the latter occupying about one-third of the area of the cross-section. Though the elements from the VII and V nerves are from this point on intimately united and bound up in the same sheath, yet in good preparations it is possible to follow them independently for a considerable distance, for they are separated by a delicate connective tissue septum. All the coarser and some of the fine fibres from the trigeminus gradually swing around mesally from the dorsal to the ventral side of the mixed trunk, while the remainder of the trigeminal fibres retain the dorsal position. A connective tissue septum appears between these two portions of the trigeminal element and finally (100) the ventral portion separates from the trunk. While it is impossible to be certain that no fibres from the facialis enter this branch, the appearances are against it and it is certain that most of its fibres are derived from the trigeminal element.

It turns toward the median line, enters the m. genio-hyoideus and then turns caudad in the substance of the muscle (*m. ghy.*), which it supplies for almost its entire length. Within the muscle it anastomoses with its fellow of the opposite side. Some fibres also emerge upon the surface under the skin of the copula and are, I think, unquestionably of general cutaneous nature.

Within the m. genio-hyoideus a twig of very coarse fibres passes to the extreme ventral surface, then cephalad near the median line (*m. im.*). Having reached the level of the m. intermandibularis, it goes dorsad and enters the middle of that muscle spreading out among its fibres in the manner typical for motor nerves. On the opposite side of this specimen this twig pursues a similar course except for the fact that it separates from the motor fibres destined for the m. genio-hyoideus before they have entered that muscle. They then run inward along the dorsal instead of the ventral surface of the genio-hyoideus and enter the m. intermandibularis at about the same place as those of the other side.

The relations of this anastomosis from the trigeminus for the mm. genio-hyoideus and intermandibularis and the adjacent skin were traced on the opposite side of this specimen and on both sides of several others and in all cases they were as above described. Osmic acid preparations (mounted unstained after fixation in Hermann's fluid) show with especial distinctness that this nerve arises from the trigeminus and not from the facialis.

The remaining fibres of this communicating branch from the trigeminus after the separation of the branch last described, join the r. mandibularis externus VII and from the facialis portion of the mixed trunk thus formed, there arises at once a small twig (*m. VII. 12*) for the third

mandibular canal organ. Then under the origin of the m. intermandibularis the trunk divides into two nearly equal portions of which the dorsal one contains all of the coarse, *i. e.*, facialis fibres. These supply the second and first mandibular canal organs, the remaining or trigeminus fibres supply the skin of the tip of the mandible and the middle portion (not the edges) of the lower lip.

5.—Comparative Review of the Hyomandibularis.

The truncus hyomandibularis receives fibres belonging to the viscero-motor and the three sensory components. The general cutaneous component is very small. In Lota and Esox (Goronowitsch, '96) the same relations prevail, though in Lota (p. 28 and Fig. 12) the general cutaneous portion is very large and passes off from the Gasserian ganglion as a broad anastomosing band. It would be interesting to learn the exact distribution of these trigeminal fibres in Lota.

i.—The opercular rami.—The muscles supplied by the r. opercularis profundus are undoubtedly to be compared with the general constrictor system of the facial region of selachians. The lateralis fibres in the r. opercularis superficialis have been already discussed. The general cutaneous portion of this nerve is, I believe, peculiar to the teleosts. It is of the same nature as the cutaneous portion of the r. hyoideus.

ii.—The ramus hyoideus.—This nerve is usually described as a pure motor nerve, and such it probably is in most vertebrates other than the bony fishes. The muscles supplied in Menidia (hyo-hyoideus) evidently belong to the constrictor system of the facialis segment and no other.

All of the general cutaneous fibres which enter the

truncus hyomandibularis are given off to the operculum through the r. opercularis superficialis and the r. hyoideus. Goronowitsch states ('96) that these cutaneous fibres are not present in Ganoids, though from the descriptions and figures of Allis ('97) it is probable that they are present in Amia. Goronowitsch accounts for their presence in teleosts on the supposition that the suspensory apparatus has been pushed forward into the territory innervated by the trigeminus. This supposition may be correct, and yet the prime motive for the entrance of fibres from the Gasserian ganglion into the truncus hyomandibularis is to be sought rather in the backward growth from the hyoid arch of the opercular apparatus. The way in which these fibres enter the hyomandibularis, as well as their peripheral distribution, go to show that they have been cenogenetically carried back by the growth of the operculum. The cutaneous twigs from the trigeminus which overlie the opercular canal and the preopercular bone (the infraorbital branch *io.* 2) have apparently also been involved in this backward movement. The skin overlying the cephalic end of the preopercular bone is not, however, innervated from these hyomandibular fibres, but from recurrent twigs from the r. mandibularis V, viz., from the anastomosing branch *V–VII.* 1. We have as yet no definite knowledge of any case where the skin of the facial region is innervated from the facial roots. The general cutaneous fibres which Strong describes as joining the truncus hyomandibularis of the tadpole from the ganglionic complex of the IX and X nerves and distributing with the r. hyoideus and the r. mandibularis externus are most puzzling, especially in view of the fact that the operculum of the tadpole is known to grow back over the gills very much as in the fishes.

iii.—The ramus mandibularis.—The course of the proximal portion of the r. mandibularis VII after its separation from the r. hyoideus as above described, differs greatly from that of the forms described by Stannius, a difference to be explained, perhaps, by the excessive development of the preoperculum in Menidia. The temporary separation of this ramus into two portions (viz., the r. mandibularis externus and the r. mandibularis internus) before entering the mandible, is a common character in the bony fishes, as appears from the works of Stannius ('49, p. 63) and Vetter ('78, p. 479). In Cottus alone the two portions do not re-unite (Stannius).

In my preliminary paper ('97) I described the naked organs along the opercular canal as innervated from the communis component. In this I was unduly influenced by the size of the fibres. These nerve fibres are scarcely larger than those which supply terminal buds on the top of the head, yet they are very heavily myelinated so that they stain very intensely. I have since that time traced them with great care in a number of specimens and am convinced that in every case they arise from the lateralis component (r. mandibularis externus) and not from the communis (r. man. internus). The organs are, I believe, to be regarded as more or less degenerate pit-lines. They are smaller than the similar organs supplied by the r. opercularis superficialis and this may account for the fact that the nerve fibres supplying them are of smaller size.

I agree with Ruge ('97, p. 216) that the peripheral relations of the facial nerve contribute nothing to the hypothesis that a branchiomere was originally intercallated between the hyoid and the mandible. If such a segment ever existed, it has left as small trace in the adult peripheral nervous system as in the central. Neal ('98) has

shown in Acanthias that a neuromere is found in this position in the brain, but it is early lost, leaving no trace behind. We cannot, then, reasonably look for such a segment in the adult of the more highly specialized teleost.

iv.—The genio-hyoideus and intermandibularis muscles.— Previous investigators have been unable to determine the innervation of these muscles with certainty, as the methods of dissection are obviously inadequate to unravel the anastomoses between the VII and V nerves.

Stannius mentions (p. 23) that the m. genio-hyoideus in some cases contracts when the motor V (his first root) is stimulated; nevertheless he is inclined to regard the proper innervation of the muscle from the facialis. He also found (p. 62) that in Esox stimulation of the truncus hyomandibularis causes movements of the branchiostegal membrane and also weak movements of the lower jaw, and this would favor that view.

Vetter ('78, p. 515) found that in this species the m. genio-hyoideus is supplied largely by extensions of the r. hyoideus which run forward into it after supplying the branchiostegal muscles (m. hyo-hyoideus). This is not mentioned by Stannius and certainly is not true in Menidia, yet I may account for the movements of the jaw observed by Stannius upon stimulation of the truncus hyomandibularis.

Subsequent studies (July, 1899) have shown me that this is, however, true in Gadus. Or at any rate the r. hyoideus runs forward from the branchiostegal membrane in this species to anastomose with the nerve for the genio-hyoideus within the substance of that muscle. The figure and description by Allis ('97, p. 613 and Fig. 43) indicate a similar condition for Amia.

Pending an exact embryological examination, the morphology of the pre-hyal ventral musculature of the teleosts has remained obscure. It is supplied, at least in part, by

the spinal nerves or the spino-occipital nerves (including the hypoglossus) in all other vertebrates. See Fürbringer ('97) for details and literature. The isolation of the teleosts in this respect is striking.

In selachians (Vetter, '78), in Ceratodus (Ruge, '97) and in ganoids (Allis, '97) we have in the pre-hyal region in addition to the longitudinal spinal musculature, a general ventral constrictor system supplied by the V and VII nerves. Now, the mm. intermandibularis, genio-hyoideus and hyo-hyoideus of ganoids and teleosts have unquestionably been derived from this ventral constrictor system of selachians, as has been shown by Vetter ('78), Ruge ('97) and with especial clearness by Allis ('97, p. 582, seq.)

In the teleosts it may safely be asserted that the pre-hyal "hypoglossus musculature," which in other forms is supplied by the first spinal or by the hypoglossus and which is known to grow forward from the post-otic myotomes, is altogether absent. I think that future embryological studies will confirm this and the condition is probably to be explained by the overgrowth of the opercula and the peculiar conformation of the isthmus.

The so-called genio-glossus muscle of teleosts would, then, not be homologous with the muscle of that name in most other vertebrates. It is, however, homologous with that muscle in the ganoids, for both have been derived from the constrictor system and are innervated from the V and VII nerves. The m. branchio-mandibularis of the ganoids is a true pre-hyal ventral muscle and it is accordingly innervated from the spinals, and all authors agree that this muscle is not present in the teleosts. In the bony ganoid Amia, which shows so many other teleostean characters, it is significant that we find the branchio-mandibularis effecting its insertion only in late larval or

adult life and then in so exceedingly variable a manner as to suggest that the muscle is in a process of degenerescence (Allis, '97, p. 700). In this fish the genio-hyoideus and the intermandibularis muscles are clearly supplied from the V, and the trigeminal nerves which supply them (*r. ghs.* and *r. ghi.*) are evidently comparable with my anastomosing branch *V–VII.* 2. Like the latter they contain general cutaneous fibres and Allis thinks ('97, p. 638) also communis fibres for the terminal buds found on the gular plate and the lower end of the gill cover in Amia. In Lota, too, (Goronowitsch, '96, p. 40) the intermandibularis is clearly innervated from the trigeminus (his Trig. I).

In forms like Esox, in which the so-called genio-glossus is innervated from both the VII and V nerves (Vetter '78), we may assume that the muscle represents both facial and trigeminal constrictor systems, comparable with those of Ceratodus, while in Menidia and most other teleosts the VII portion has been lost and the m. "genio-hyoideus," together with the intermandibularis, represents the ventral constrictor muscles of the trigeminus segment, the facialis constrictor muscles being represented only by certain dorsal opercular muscles and by the branchiostegal muscles.

Ruge ('97) is not willing to accept this interpretation; but, being convinced that these muscles must in all forms belong to the facial segment, he makes the entirely gratuitous assumption that the motor fibres for these two muscles, which in Ceratodus and teleosts appear to come from the trigeminus, really come as anastomosing fibres from the facial.

This assumption he makes also for the innervation of the intermandibular muscle of Amphibia, Reptilia and

Mammalia, and since in the two latter cases there are no peripheral anastomoses between the rami of the VII and V nerves, he is obliged to postulate "an intra-cranial fusion of the two nerves," the very existence of such an anastomosis being a pure assumption.

In Menidia, at any rate, it is clear that the exact reverse is true, the apparent innervation from the facial in reality being derived from the motor nucleus of the trigeminus, as there is no possible opportunity for a confusion at any point between motor fibres of the V and VII nerves.

v.—The chorda tympani.—It may be regarded as established that the chorda tympani in man supplies taste buds on the anterior part of the tongue and glands and also perhaps general mucous surfaces at the base of the tongue and between it and the lower jaw. Its nerve fibres arise from the geniculate ganglion of the facial and enter the brain through the portio intermedia of Wrisberg, and internally they are said to pass to the IX nucleus by way of the fasciculus solitarius. Now, the fasciculus solitarius we homologize with the fasciculus communis of the Ichthyopsida and hence it appears that the chorda both centrally and peripherally is a visceral branch of the facialis. That this nerve is a pre-trematic branch is indicated by the peculiar course of the chorda, through the tympanic cavity and above and in front of the Eustachean tube, the latter being regarded as the homologue of the spiracle.

These homologies cannot be regarded as definitely established; nevertheless they are greatly strengthened by the direct embryological evidence brought out by Dixon's work, On the Development of the Branches of the Fifth Cranial Nerve in Man ('96). This author found that the chorda tympani and large superficial petrosal

(Vidian) nerves grow out from the seventh nerve, undoubtedly from the geniculate ganglion, and that as late as the fifth week the chorda had not effected its connection with the lingual nerve. These results are of themselves quite sufficient to confute such work as that of Penzo ('93), who tries to prove *by dissection* that the chorda tympani and great superficial petrosal nerves contain fibres derived from the trigeminus.

Lenhossék ('94) found that fibres from the cells of the geniculate ganglion enter the portio intermedia, but that the fibres of the large superficial petrosal are not connected with the geniculate ganglion cells. But this negative result cannot stand in the face of the embryological data of Dixon and the degeneration experiments of Amabilino ('98). The latter author got a characteristic Nissl degeneration of the cells of the geniculate ganglion after destruction of the chorda, but no degeneration after resection of the facialis. He demonstrated the degeneration of these fibres peripherally of the lesion and also found that some chorda fibres do not degenerate. It is possibly these ascending fibres which Lenhossék found not to terminate in the ganglion. Amabilino's work has been since confirmed by Van Gehuchten (*Journal de Neurologie, 1898*), with, however, the important difference that Van Gehuchten found that section of the facial nerve of the rabbit immediately after its emergence from the Fallopian canal does cause degeneration of a few cells of the geniculate ganglion, thus showing that the facial nerve receives some communis fibres.

The most important evidence against this conception of the chorda comes from the clinical side. Compare especially the case cited by Adolf Schmidt ('95), which proves conclusively that sensations of taste are transmitted

from the anterior two-thirds of the tongue by means of the lingual nerve (V+VII), rather than by the IX nerve. Clinical evidence is then adduced to show that these gustatory fibres enter the brain through the V nerve rather than the VII, which, however, does not seem to me by any means conclusive.

A pre-facial fasciculus solitarius has been described in man by a few writers (Böttiger, '90, and Roller, '81). Only upon the supposition that such a tract does enter the trigeminus (a condition which has not been demonstrated as yet in any of the lower animals) could the presence of gustatory fibres in the trigeminus roots be explained. In the present state of our knowledge we may most safely consider that Dixon's conclusion in man applies to all of the vertebrates: "The nerve supply of the organs of taste appears to be derived from the facial and glossopharyngeal nerves alone."

In man, then, so far as is definitely known, the pre-auditory communis system is represented by the large superficial petrosal nerve and the chorda tympani, plus a few fibres in the facial proper—see the reference to Van Gehuchten above. Since the days of Stannius the large superficial petrosal is pretty generally regarded as the homologue of the r. palatinus. The homologies of the chorda tympani, however, have given more trouble. In determining this question there are three criteria or lines of evidence which have been very differently estimated by different authors: (1) The character of the fibres, their ganglion and central termination, (2) the peripheral distribution area, (3) the intermediate course of the nerve, especially with reference to the spiracle and its limiting arches.

Froriep's comparison ('87) with a lateral line nerve of

course fails because it contravenes the first criterion. The first and second criteria are perfectly fulfilled by the mandibularis internus VII of the Amphibia (Strong, '95) and by that nerve of the fishes in general, for it supplies the mucosa between the hyoid and mandibular arches and along the inner side of the latter (see also Ruge, '97, p. 209).

Several recent writers in emphasizing the third criterion above have called attention to the fact that the r. mandibularis internus VII of several of the fishes is a post-spiracular nerve and therefore cannot be homologous with the chorda tympani. This point was made by Allis in his preliminary paper in 1895 (p. 488), but his discussion of this nerve in his later paper ('97, p. 638) is not altogether clear. He distinctly homologizes this nerve in Amia with the nerve so named by Ewart, Pollard and Strong in the other Ichthyopsida, for it certainly has the corresponding course for its whole length. Since it appears to take no part in the innervation either of terminal buds externally or of taste buds in the mouth, he seems inclined, however, to conclude that it is not a communis nerve at all, but perhaps general cutaneous. This he bases on the false assumption elsewhere expressed (p. 642), that all, or nearly all, communis fibres are for specialized sense organs. The fact is that they may go to the general mucous surfaces, and I have no doubt that the r. mandibularis internus VII of Amia is structurally, as well as topographically, the same as that nerve in Menidia and the other forms mentioned. As this nerve in Amia and in selachians lies behind the spiracular canal, the corresponding nerve in these other types must also be a real post-trematic ramus and cannot be regarded as a pre-trematic ramus which has coalesced with the post-

trematic nerve (r. hyomandibularis) after the occlusion of the spiracular canal.

The r. mandibularis internus VII of the Ichthyopsida, then, probably does not correspond to the chorda tympani of the higher forms, but it is a primary component of the post-spiracular nerve of vertebrates in general. When absent from that nerve (as sometimes occurs) this fact, rather than its presence, is to be explained as a secondary modification.

The relations of the pre-spiracular communis nerves are exceedingly diverse in different vertebrates. The primary arrangement was doubtless the typical palatine and pre-trematic branches, but even in the selachians there is considerable deviation from the type in various directions. As differentiation progressed, the development of taste buds on the lips (correlated with the absence of communis fibres in the trigeminus) led not only to the extension of the typical facial nerves (palatine, pre-trematic and post-trematic) from their proper arches to supply them, but in some cases to the formation of entirely new nerves, such as the internal rostral nerve of Acipenser, the communis component of the r. maxillaris of other fishes and, as we shall see below, the chorda tympani. The later development of a fleshy tongue has led in higher vertebrates to a similar prolongation of one or more branchial nerves to innervate it.

Now, the known diversity, even in rather closely related fishes, in the nerves which are thus prolonged should incline to the greatest caution in establishing homologies, especially those based upon peripheral relations. Thus, any of four distinct communis nerves may run forward upon the hyoid and mandibular arches:—(1) the r. mandibularis internus VII, (2) the r. pre-trematicus VII,

(3) communis fibres secondarily added to the r. mandibularis V, or (4) a nerve distinct from any of the preceding. Which of these nerves persists in a given case after the obliteration of the spiracle is a matter which it may not be possible to determine from the adult peripheral relations, nor is it safe to assume that it is the same nerve in all cases.

The condition in Raja and Spinax is especially fruitful in suggestions for the interpretation of higher forms. Here Stannius describes ('49, p. 57) three branches of the r. palatinus:

1. A small posterior (caudal) branch, which supplies the pseudobranch of the spiracle. This is manifestly the proper r. pre-trematicus VII, *i. e.*, the nerve for the pre-spiracular demibranch.

2. The first anterior branch. A stronger nerve which sends a twig back for the anterior lining of the spiracle, anastomoses with branches 1 and 3, and then runs forward and inward under the mucous lining of the mouth between the hyoid and the mandible, reaching to the ventral median line. This nerve perfectly fulfils every condition for the chorda tympani, *and is an independent nerve for its entire length.*

3. There is, finally, the second anterior branch, or true r. palatinus.

We have in these fishes, then, a chorda tympani *in addition to* the r. palatinus, the pre-trematic VII and the post-trematic VII.

Chimæra would seem from Cole's description ('96a, p. 652) to present a simple and primitive condition. The r. pre-trematicus VII arises from the base of the palatine and distributes to the ventral portion of the pharynx behind the lower jaw. This he calls (I think correctly) the

chorda tympani. The r. pre-trematicus in the strict sense (number 1 of Stannius' description) may have fused with this nerve, or more likely has disappeared with its demibranch. Midway of its course it receives an anastomosing branch from the facial proper (post-spiracular) and this is probably the post-trematic communis element of the facial, secondarily and incompletely joined to the pre-trematic ramus—a conclusion suggested to me by Mr. Cole in private correspondence.

An anastomosis of the type presented in Chimæra is unusual. More often it is the r. pre-trematicus and chorda which lose their identity after the closure of the spiracle, either by fusion with the post-trematic facial ("facial proper" of Ewart and Cole) or by fusion with the post-trematic trigeminus (r. mandibularis V). The latter type is the one which would most naturally occur, since both of the anastomosing nerves run along the same arch; and, in fact, it is apparently most frequently found among the fishes. It does *not*, however, appear in Menidia; for this fish, as we shall see beyond, lacks communis fibres in the r. mandibularis V, though such fibres occur in the r. maxillaris. But Menidia does possess a distinct nerve (to be described in the next sub-section) between the truncus hyomandibularis and the trigeminus which distributes to the region of the pseudobranch and which I regard as the r. pre-trematicus VII (in the narrow sense).

In Amia, Allis finds communis fibres in both the maxillary and the mandibular rami of the trigeminus. These fibres come "from the ganglion of the fasciculus communis root" ('95, p. 488), *i. e.*, the geniculate ganglion, as in Menidia. Since there are no communis (*i. e.*, pre-spiracular VII) fibres in the r. mandibularis V in Menidia,

the mucous covering of the upper end of the hyoid arch is supplied from the r. mandibularis internus VII (my branch *m. VII. 3*), *i. e.*, from the post-spiracular VII. Such fibres do occur in the r. mandibularis V of Amia and accordingly we find the taste buds and mucosa of the corresponding region supplied by the fourth branch of the mandibularis V ('97, p. 612). Allis is, doubtless, correct in homologizing these fibres with the chorda tympani, *i. e.*, they correspond to Stannius' branch 2 of selachians. The r. pre-trematicus *sensu stricto* (branch 1) may also be represented in this nerve, but more probably in the "posterior palatine" of Allis (see below).

Goronowitsch ('96) has also described, though without recognizing their significance, similar fibres from the geniculate ganglion to the r. mandibularis V in Lota vulgaris. This I can confirm (July, 1899) in Gadus morrhua, and can add that here, as in Amia, the mucosa of the hyoid region is supplied by these mandibular fibres, and not by the r. mandibularis internus VII.

I would suggest that in Menidia the independence of the r. pre-trematicus VII is to be accounted for by the great size of the pseudobranch, which is innervated by this nerve. Not having fused with the r. mandibularis V, the pre-trematic VII is distributed only to the area about the pseudobranch; and the regions farther cephalad, along the hyoid and mandibular arches, which are supplied by other pre-trematic fibres in some other forms, in Menidia are supplied by the post-trematic branch. Menidia clearly lacks the chorda in the proper sense of that term, the post-trematic communis element replacing it functionally.

I am inclined, therefore, to regard the nerve to be described beyond as the r. pre-trematicus VII of Menidia as the equivalent of Stannius' branch 1 of selachians. It is not properly the homologue of the chorda tympani of mam-

mals, for it does not conform to the third criterion mentioned above. That is, it does not fuse with the r. mandibularis V and hence it does not distribute to the hyoid and mandibular arches in the way characteristic of the mammals.

Cole, in his discussion of the chorda already referred to ('96a, p. 657 ff.), gives a vigorous and I think conclusive argument for the pre-spiracular nature of the chorda, but his homologies in the several groups of fishes and amphibians are open to criticism in several respects.

In the first place, he is misled by a false conception of a typical branchial nerve. The pre-trematic ramus he regards as sensory and the post-trematic as "practically motor." The latter point is incorrect; the post-trematic ramus is typically mixed, and the presence of communis fibres (r. mandibularis internus VII) in the post-trematic VII is strictly typical. (This is true also even in the Mammalia, if Van Gehuchten's most recent work, cited above, is sound.) He homologizes his chorda with Strong's internal mandibular, but considers that the latter is a pre-trematic nerve which has fused secondarily with the post-trematic. He adds: "My reasons for this assertion are two—(1) his man. int. arises *from the base of the palatine*, which is almost invariably the origin of the pre-spiracular nerve; (2) it consists entirely (?) of splanchnic sensory fibres (and thus agrees with the palatine), whereas the post-spiracular division of the VIIth is practically motor."

Now, the first of these reasons loses its force entirely when we remember that nearly all of the communis fibres of the VII nerve diverge into their respective rami immediately upon leaving the ventral edge of the geniculate ganglion, so that both the pre-trematic and the post-

trematic branches must of necessity arise "from the base of the palatine." Compare especially my Fig. 4. His second reason we have criticised just above.

Strong's internal mandibular is, I am confident, the same nerve as the one so named in Menidia, and I believe it to be homologous with the post-spiracular mandibularis internus of Amia (Allis, '97) and of some selachians (Stannius, '49, p. 65, and Jackson and Clarke, '76), and not completely so with the chorda of mammals. I say not completely, for it may well be that the pre-trematic VII in the frog has fused with the post-trematic, or hyomandibular nerve, so that the amphibian r. mandibularis internus VII may represent both the pre- and the post-spiracular communis elements.

Cole, in his admirable memoir on the nerves of the cod-fish ('98a), re-states his argument on the chorda (pp. 200–201). Though no new facts are brought out, yet the "confusion in the terminology of the facial nerve of fishes" is, I fear, rather augmented than diminished, and an examination of his argument in detail is necessary.

In the first place he says: "As I have already pointed out (1896, *46*, p. 657 *et seq.*) *the terms internal mandibular and hyoideus as first used by Stannius are not only synonymous but apply to a motor post-spiracular* nerve related to the anterior face of the hyoid arch, just as the pre-spiracular nerve should be related to the posterior face of the mandibular arch." This, I think, is hardly fair to Stannius.

In selachians Stannius describes ('49, p. 65) two branches of his (post-spiracular) truncus hyoideo-mandibularis, the r. mandibularis externus and the r. mandibularis internus s. profundus, which have since that time been very generally regarded as lateralis and visceral nerves respectively, the latter distributing to the mucous lining of the mouth. In his description of the teleostean arrangement both of these components of the r. mandib-

ularis VII are described, though the names external and internal are not applied to them, and these are *in addition* to the r. hyoideus. On pp. 62–63 he describes motor and general cutaneous elements in the r. hyoideus, exactly as in Menidia, and in some cases also branches to the lateral line organs of the operculum. (In the latter cases it is obvious that the nerve includes also my r. opercularis superficialis VII). He then proceeds to describe the r. mandibularis, as follows:

,, Dieser Ast, bald schwächer, bald stärker als der vorige, verläuft bei den Knochenfischen an der Aussenfläche des Os temporale, bedeckt vom Schläfenmuskel, etwas vorwärts, gibt gewöhnlich einen oberflächlichen, zum Os quatrato-jugale sich erstreckenden Zweig A [r. mandibularis internus VII of Menidia, etc.] ab, tritt dann in einen Canal des Os tympanicum und gelangt aus demselben an das Os symplecticum, um längs demselben zum Unterkiefergelenke zu treten. Hier nimt er [*i. e.*, the r. mandibularis externus] gewöhnlich—namentlich bei Cyclopterus, Belone, Gadus, Pleuronectes, Salmo, Coregonus—den zuerst abgetretenen grösseren Zweig A, nachdem dieser das Os quadrato-jugale durchbohrt hat, in seine Bahn wieder auf; seltener, wie z. B. bei Cottus, bleibt dieser Zweig A vom Stamme gesondert, und vertheilt sich am Boden der Mundhöhle unter der Schleimhaut.

,, Der Stamm des Ramus mandibularis erstreckt sich dann an der Innenfläche des Unterkiefers, unter dem Meckel'schen Knorpel, in der diesen aufnehmenden Längsrinne vorwärts bis zur Verbindung beider Unterkieferhälften. Er vertheilt sich, nach eingegangenen Verbindungen mit dem R. maxillaris inferior N. trigemini,—bei Lophius findet eine doppelte Verbindung dieser Art Statt [as in Menidia]—in den die beiden Unterkieferhälften an einander ziehenden Muskel, in den Musculus geniohyoideus, an der Schleimhaut des Mundes und an der den Unterkiefer bekleidenden äusseren Haut."

My account in Menidia, it will be observed, conforms exactly to this description; and the branch A (especially in Cottus where its peripheral course is distinct from the

rest of the nerve) clearly is a visceral nerve for the mucosa of the mouth, the r. mandibularis internus VII of Stannius in the selachians and of Strong for the Amphibia. In Stannius' discussion of the homologue in the selachians of the r. hyoideus of teleosts (p. 65) I find no statement which would identify the "internal mandibular" with a motor nerve in either selachians or teleosts, and there is no reason why that term should not be applied to the communis component of the r. mandibularis VII of the bony fishes as in other vertebrates.

Cole follows (p. 202) with a tabular presentation of his conception of the composition of the facial nerve, a conception which I think inadmissible in several particulars. My own view of the branchiomeric characters of the facial nerve is given under the caption *Metamerism* in Section 12.

As these sheets pass through the press (July, 1899), I am constrained to add a further note. Cole has admitted the misquotation of Stannius, both to me privately, and later publicly in the *Anatomischer Anzeiger* (XVI, 2, 1899, p. 40, ff.) Since the matter is of some morphological importance, I have, however, left the criticism as originally written, in order that Stannius' own words might be before us.

Another matter may be touched upon here. In the course of a microscopical review of the trigemino-facial complex of Gadus morrhua, upon which I am now engaged, I find that the post-spiracular communis element of the facialis is totally wanting (or at most, so reduced as to be unrecognizable), *i. e.*, there is no r. mandibularis internus VII in Gadus (Cole's statement to the contrary, '98a, p. 202, notwithstanding). The nerve which I have called the r. pretrematicus VII is present in exactly the same relations as in Menidia, and there is *in addition* a large communis element in both the r. maxillaris and the r. mandibularis V. The mucosa in the region of the suspensorium and of the mandible which is supplied from the r. mandibularis internus VII in Menidia is supplied from the mandibularis trigemini in Gadus.

In the light of the preceding discussion it would therefore appear probable that in Gadus the r. pre-trematicus VII represents the nerve 1 of Stannius in selachians and that the nerve 2 (*i. e.*, the chorda tympani) has fused with the r. mandibularis V. In any case this emphasizes the difficulty alluded to above of defining the homologies of these nerves by topographical relations alone. It also makes

necessary some qualification of Cole's remark ('98a, p. 200): "There are several cases on record where on the disappearance of the spiracle the pre-spiracular accompanies for a time the post-spiracular nerve and thus becomes a topographical, but not a morphological, post-spiracular nerve. It seems to me that when the early development of the nerves of *Amia* has been investigated it will be found that the 'internal mandibular' nerve is morphologically pre-spiracular, though occupying a post-spiracular position in the adult. This is what we know has happened in *Rana* (cp. Strong's 'internal mandibular'), and what has doubtless also happened in *Chimæra* and *Gadus*." This, it seems to me, simply begs the question. There are no facts, so far as my knowledge goes, which would permit us to say that we "know" of any such secondary changes in the relations of this nerve, though such changes are theoretically possible.

In the case of Amia, Allis has replied (*Anat. Anzeiger*, XV, p. 374), stating specifically that the r. mandibularis internus VII is post-spiracular in the larval Amia. In the case of Chimæra, we concluded above that the post-trematic communis element has fused with the pre-trematic, rather than the post-trematic ramus. And in Gadus the fusion of the pre-trematic communis element with the post-trematic is manifestly impossible, for the simple reason that the post-trematic trunk totally lacks communis fibres.

The r. pre-trematicus VII of my description is unquestionably the same nerve as the posterior palatine nerve of Gadus (Cole, '98a, p. 135). It is not so certain that it is the same as Allis' posterior palatine of Amia ('97, p. 619). It has the same origin and it runs out in front of the pseudobranch, but its distribution seems to be far cephalad and laterally along the border of the maxilla. If it supplies the pseudobranch, then this part would be homologous with the nerve in question. The remainder of the nerve cannot be compared with anything in Menidia.

In Protopterus the r. mandibularis internus VII is evidently from the description of Pinkus ('94) in part, at least, the post-trematic communis element, as in other Ichthyopsida, and not a pure motor nerve, as Cole supposes ('98a, p. 201). Pinkus regards his r. palatinus inferior as the chorda tympani and homologizes it (as Miss Platt does the "external palatine" of Necturus, '96, p. 534) with Strong's r. mandibularis internus VII. The latter of these conclusions is doubtless incorrect, for Strong's nerve is, as we have just seen, also present. Allis may be right in identifying Pinkus' nerve with certain communis branches of the r. mandibularis V of Amia. But these relations must remain hypothetical until we have more exact knowledge of the components in Protopterus.

V.—The Ramus Pre-trematicus Facialis.

A large bundle of communis fibres runs from the ventral surface of the geniculate ganglion, enters the same foramen as the truncus hyomandibularis, crosses the latter nerve and gives to it a considerable communis component, as already described. Immediately after its emergence from the cranium it divides into two approximately equal portions; one, the r. palatinus, passes cephalad along the cranial wall under the origin of the m. adductor arcus palatini, the other (to which I have applied the name r. pre-trematicus VII, *r. VII. p. t.*) turns directly ventrad along the caudal and inner face of that muscle and between it and the large pseudobranch, whose cephalic end is crowded far dorsad (Fig 2). This nerve is chiefly distributed to the mucosa of the roof of the mouth and its contained taste buds cephalad of this point; but several large branches run caudad along the anterior surface of the pseudobranch and between its lobes. Having reached its caudal and ventral surfaces, they spread out and doubtless supply the numerous taste buds of the underlying mucosa and also the pseudobranch itself. Though these fibres on account of their extreme tenuity and delicate myelination could not be traced into the substance of the pseudobranch, yet there can be no doubt that they do innervate this organ, as they spread freely over its surfaces, and besides the pseudobranch receives no nerve supply from any other source.

The morphology of the teleostean pseudobranch, in spite of several recent papers, is in a very unsatisfactory state, and yet it is of great importance for the proper interpretation of all of the branches of the facial nerve.

Stannius states that it is usually in the teleosts innervated from the IX nerve, and cites the following cases:

Cottus, Cyclopterus, Gadus, Tincta, Salmo, Alosa, Clupea ('49, p. 77). In Belone and Esox he gives its innervation from the r. palatinus VII (his r. palatinus V, p. 56).

It is clear that in these two cases either the pseudobranch is not homologous or that a very remarkable secondary shifting of nerve connections has taken place. Now, the teleostean pseudobranch may be conceived of as the vestige of any one of three demibranches of a lower form:

(1) The hyoid demibranch in the first gill cleft.

(2) The hyoid demibranch in the spiracular cleft, *i. e.*, the cephalic demibranch of the hyoid arch, or

(3) The mandibular demibranch of the spiracular cleft.

The first would be supplied by the pre-trematic IX nerve, the second by the post-trematic VII (*i. e.*, the hyomandibular or hyoideus, the third by a pre-trematic VII nerve.

The older writers assumed that the pseudobranch in teleosts is a hyoidean gill, presumably the more caudal demibranch, though the latter point is not usually made plain. See particularly the paper by Maurer ('84). But in a second paper Maurer ('88) traced the embryology of the arterial arches in the salmon and found that it is not the first (mandibular), but the the second (hyoid) arterial arch that atrophies. The pseudobranch therefore develops, we infer, in connection with the mandibular arch and is a mandibular demibranch of the spiracular cleft. This is further supported by the fact that in the forms in which the pseudobranch is said by Stannius to be innervated trom the VII nerve (Belone, Esox) its fibres come from the r. palatinus, which is supposed to be pre-spiracular, rather than from the r. hyomandibularis, supposed to be post-spiracular.

But if this argument from the innervation were followed up, it would lead to the conclusion that in those species which have the pseudobranch supplied by the IX nerve it must be derived from the second hyoid demibranch, while in fact Stannius states that the salmon itself, whose pseudobranch Maurer decided is spiracular, has that organ innervated from the IX instead of the VII nerve. If, then, Stannius is correct regarding the innervation in this species, it follows that either the nerves or the arterial arches have suffered profound secondary modification. And we are not yet in a position to decide between these alternatives—certainly not until Stannius' account of the innervation has been confirmed microscopically.

From the neurological data now in hand it would appear that the pseudobranch of bony fishes is sometimes a vestige of a demibranch of the first gill cleft, sometimes of the spiracular cleft. It must be admitted, however, that this mode of procedure may also lead into difficulties, as *e. g.*, in Lepidosteus (Wright, '85), where the spiracular pseudobranch *seems* to be innervated by the IX nerve and the hyoidean gill by the VII. But this case requires further investigation, as all admit.

At all events, pending further study, the case of Menidia can be interpreted on the basis of the nerve supply in only one way. We naturally assume that the pseudobranch represents a mandibular demibranch of a vanished spiracular cleft, and that the nerve supplying it and the roof of the pharynx adjacent between the areas supplied by the IX and palatine nerves is a true pre-trematic VII nerve, such as is mentioned, *e. g.* by Stannius and by Ruge ('97), in some sharks. This accords with the embryological data in the case of the vascular arches of the salmon and Lepidosteus. Müller ('97) has studied the

development of the vascular arches in Lepidosteus and concludes that (in spite of the anomalous innervation) the pseudobranch is a spiracular (mandibular) gill, while the "hyoid gill" is the demibranch of the anterior wall of the first gill cleft.

I am inclined to doubt the participation of the IX nerve in the innervation of the teleostean pseudobranch in any case. In Gadus I am sure that its nerve supply comes from the facialis.

For the morphological discussion of this nerve see the pages immediately preceding.

VI.—The Ramus Palatinus Facialis.

After its separation from the nerve last described, the r. palatinus runs forward along the dorsal surface of the m. adductor arcus palatini and beyond the cephalic edge of that muscle (400) reaches the mucous lining of the roof of the mouth near the median line, which it follows up to the tip of the snout. This epithelium is richly supplied with taste buds and gland cells during almost the whole of this course and both of these are supplied by this nerve.

Stannius states (p. 55) that in fishes which have a well developed sub-cranial canal ("Augenmuskelcanal") the r. palatinus traverses it on the way to the roof of the mouth. This certainly does not apply in the case of Menidia. The sub-cranial canal is well developed, but the r. palatinus does not enter it, but runs along the outer side of the canal, not the inner, as Stannius describes in his types.

VII.—The Truncus Infra-Orbitalis.

This trunk (*t. inf.*) contains the following nerves: the r. mandibularis V, the r. maxillaris V and the r. buccalis VII. It receives the following components: the general

cutaneous, the motor V, the acustico-lateralis, the communis and a large sympathetic element. The communis element also may in some forms, as the sturgeon, go out as a separate nerve.

These components leave the ganglionic complex in a single compact trunk passing laterad, ventrad and slightly caudad from the ganglia. The V + VII ganglionic complex is all intra-cranial except the Gasserian ganglion. At about 500 (Fig. 22) the complex becomes narrower, as all of the VII ganglia lie caudad of this point, while most of the Gasserian ganglion lies cephalad; and here the whole of the complex (with the exception of the truncus hyomandibularis, the palatine and pre-trematic rami and one root of the r. lateralis accessorius, which have been previously given off) turns abruptly outward and emerges from the cranium through a single foramen and continues cephalad along the outer surface of the cranial wall.

The general cutaneous fibres arise from about the middle of the ventral side of the Gasserian ganglion. From the extreme caudal edge of the ganglion a small fascicle of cutaneous fibres emerges through a separate foramen and joins the truncus hyomandibularis, as already described. A similar fascicle arises from the ventral edge of the Gasserian ganglion near its cephalic end (485), curves back along the mesal face of the infra-orbital trunk at its origin, follows for a short distance the r. opercularis V (*t. f.* 1, Fig. 22), then separating from that nerve continues caudad along the dorsal border of the m. adductor arcus palatini, it joins the fascicle from the caudal end of the Gasserian ganglion (Fig. 2, *t. f.* 1) and the two together enter the truncus hyomandibularis.

Of these rami communicantes n. trigemini ad n. facialem, I can find no mention of the more caudal one, while

the cephalic one conforms closely to the typical arrangement as given by Stannius (p. 47). This latter nerve was found by Stannius in all of the fishes examined by him, with the exception of Amiurus and those in which the VII emerges from the same foramen as the V. In Belone he found it to be composed chiefly of coarse fibres, with a smaller number of fine ones. I find, however, that both rami communicantes are composed exclusively of very fine fibres, though by analogy with other general cutaneous nerves I should have expected a few coarse ones to be mingled with them, and more especially as in some pure cutaneous ramuli of the r. hyoideus, which must be derived from these rami communicantes, there are numerous coarse and medium fibres. This case is typical of many others which lead me to believe that the calibre of individual fibres may vary widely in different parts of their courses.

The motor V fibres run under the Gasserian ganglion and after their emergence take a position on the caudal and inner side of the infra-orbital trunk. The acustico-lateralis component arises from the dorsal lateralis ganglion and its fibres pass out on the cephalic and lateral face of the trunk. The communis fibres from the geniculate ganglion which have not been given off intra-cranially are somewhat confused with the general cutaneous from the Gasserian ganglion. A part of these fibres go out with the roots of the r. lateralis accessorius, others go out with the supra-orbital trunk, but the larger part is clearly seen to enter the infra-orbital trunk.

The four nerves which are represented in the infra-orbital trunk are so confused, even in their smaller branches, that they cannot well be described separately. I shall, therefore, describe the branches of the trunk in

the order in which they are given off irrespective of the components contained in them. Remembering that the r. buccalis contains all of the lateralis fibres, the r. mandibularis V all of the motor and a part of the general cutaneous, and the r. maxillaris the remainder of the general cutaneous, the peripheral relations of each of these nerves can be easily gathered from the plot (Fig. 3). Before taking up the detailed account, emphasis should again be laid upon the fact that the branches of this trunk are enumerated in this way simply as a matter of convenience in description. The details of the arrangements of these branches and their fusions with each other have no especial morphological significance, but rather seem to be determined by the individual conditions as a matter of mechanical and functional adaptation. The remarkable way in which different nerves have been compacted into the infra-orbital trunk is obviously due to mechanical causes, chiefly to the excessive development of the eyes.

1.—*The R. Opercularis V.*

The first branch to be given off from the infra-orbital trunk is the motor nerve already referred to, which arises close to the cranium, passes back a short distance in company with the first recurrent twig from the Gasserian ganglion to the truncus hyomandibularis, separates from this twig, turns outward, crossing transversely the cephalic edge of the hyomandibular bone and then divides into ventral and dorsal branches (Fig. 22) for the m. levator arcus palatini (Fig. 4, *m. l. a. p.*) and the m. dilator operculi (*m. d. op.*) respectively. These motor nerves, it appears from Stannius' description of other species in which the truncus divides into its rami before their separation, should be relegated to the r. mandibularis V.

2.—The Second and Third Branches.

The trunk runs out under the orbit between the m. rectus externus and the m. adductor arcus palatini and in this position gives off a small branch of coarse and fine fibres which passes laterally and slightly ventrally to the skin overlying the m. adductor mandibulæ (*io. 2*). Here the branch breaks up into several twigs, some of fine fibres for the skin under the eye, a coarse-fibred twig for the 12th organ of the infra-orbital line, which is a naked papilla, and the remainder continues ventrad and caudad under the skin. Having reached the opercular canal, this twig enters the bony canal, having previously penetrated the m. adductor mandibulæ and the pre-opercular bone. In the canal it divides and sends branches to the skin cephalad and caudad of this point. It sends minute twigs to the skin along its entire course.

On the opposite side of the specimen figured the course of this nerve is as described above except that the opercular general cutaneous portion breaks up earlier into a number of very fine ramuli, none of which could be traced into the opercular canal. The fibres of these ramuli join the sub-dermal plexus and lose their sheaths. They evidently supply the same region as on the other side, but by a somewhat different course.

The general cutaneous fibres of this second branch correspond in nature and position rather closely to the most lateral one of the three accessory trigeminal branches which arise from the Gasserian ganglion in the tadpole of the frog (Strong, '95).

After the separation of this second branch, the infra-orbital trunk immediately divides into three divisions, all of which contain both coarse and fine fibres. The ventral

division separates first. It corresponds in part to the r. buccalis, but it contains only a portion of the buccalis fibres and it carries other fibres also not belonging to that nerve, viz., general cutaneous and motor. The middle division contains more fine than coarse fibres and is the proper r. mandibularis V. The dorsal division contains coarse and fine fibres in about equal proportions and corresponds to the r. maxillaris plus a portion of the r. buccalis and a communis element.

After this division has begun, but while the dorsal and middle divisions are still in contact, a small branch (*io. 3*) is given off from the dorsal one. It contains only coarse fibres and goes around the outer (lateral) side of the middle division but around the inner and ventral side of the ventral one. It runs over the dorsal edge of the m. adductor mandibulæ to the skin. It now sends about half of its fibres into the substance of this muscle. These are very coarse and apparently branch freely within the muscle, for the number here is greater than the number which enters the muscle. They are undoubtedly motor fibres. The remaining fibres run a short distance downward between the m. adductor mandibulæ and the skin and in this position cross (apparently without anastomosis) one of the ventrally directed twigs of the second branch of the infra-orbital trunk, the latter twig being outside. They then supply the 11th organ of the infra-orbital line, which is a naked papilla.

3.—The Ventral Division.

This assemblage of fibres turns outward under the eye and over the m. adductor mandibulæ. Here it gives off its first branch of coarse fibres (the fourth infra-orbital branch), which curves laterally around the m. adductor

mandibulæ to the skin, where it supplies the 10th lateral line organ, a naked papilla, of the infra-orbital line. Several smaller branches of fine fibres go off at nearly the same point and supply the skin adjacent, and particularly that about the lower border of the eye (fifth infra-orbital branch).

The ventral division now comes to lie at the dorso-lateral edge of the m. adductor mandibulæ close under the skin and the coarse and fine fibres, which hitherto have been mingled, become segregated, the coarse ones all lying in the lateral half of the nerve. This tendency of lateralis fibres to draw away from the other components is very characteristic, and is exhibited in nearly all nerves where they are associated with other fibres.

At 395 another coarse-fibred twig is given off for the 9th (naked) organ of the infra-orbital line (6th infra-orbital branch). At 385 the 7th ramulus goes ventrad to supply the thickened epidermis lying between the opercular canal and the eye. The 8th ramulus supplies the 8th (naked) organ of the infra-orbital line and also the similar organ (7th) next cephalad. Then follow the 9th ramulus directed ventrad, and the 10th dorsad, both for the skin. The latter crosses external to, but apparently does not anastomose with, the twig from the dorsal division for the 6th infra-orbital organ (the 18th infra-orbital branch). The remaining fibres of this ventral division (11th infra-orbital branch) are all coarse. They enter the ventral part of the m. adductor mandibulæ and apparently participate in its innervation.

4.—The Middle Division—R. Mandibularis V.

The middle division is the r. mandibularis V. After its separation from the ventral division this nerve runs parallel to and almost in contact with the dorsal division

along the ventro-lateral side of the latter for a considerable distance. Its first branch, the 12th of the infra-orbital trunk (440), is of coarse fibres, which innervate the mesal portion of the m. adductor mandibulæ cephalad of this point. The main nerve now passes farther laterally in the floor of the orbit, separating somewhat from the dorsal division. Its coarse fibres, which we shall see are all of the motor type, are mingled among the fine ones and do not segregate themselves as lateralis fibres usually do.

The 13th branch leaves the r. mandibularis V at 375. This, too, is of coarse fibres and supplies the lateral portion of the m. adductor mandibulæ. At about 300 the r. mandibularis V turns ventrally, passing between two portions of the adductor mandibulæ and finally running forward along the outer face of the ventral edge of the quadrate near its cephalic end. The r. mandibularis VII lies in a corresponding position on the inner face of the same bone a little farther ventrad.

While in this position the r. mandibularis V gives off another branch (the 14th infra-orbital, *V–VII. 1*) composed of fine and medium fibres. Beyond the cephalic end of the quadrate this branch turns outward and under the skin over the articular bone and between and behind the open ends of the infra-orbital and mandibular canals it divides into numerous ramuli. Several of the smaller ramuli supply the skin adjacent to the end of the infra-orbital canal and that about the open space between the mandibular and opercular canals (it will be remembered that the infra-orbital canal disappears for a part of its course and closes in again at this point), and a larger ramulus runs caudad under the skin along the ventral edge of the m. adductor mandibulæ. This is the com-

municating branch with the r. mandibularis VII (*m. VII. 8*) mentioned in the account of that nerve. It sends a twig at once into the muscle, which appears to anastomose with the motor twig which enters the muscle near the same point from the ventral division of the infra-orbital trunk. The fibres of this twig, like the rest of those of the communicating branch, are fine or medium and very different from typical motor fibres. The rest of this nerve runs, as before described, along the ventral surface of the m. adductor mandibulæ, becoming progressively smaller caudad, and finally joins the r. mandibularis VII. This may be regarded as a sensory nerve for the muscle or as a general cutaneous nerve for the overlying skin.

The ramulus last described receives some fibres from the r. mandibularis VII (*m. VII.* 8) and these enter the largest ramulus of this group from the 14th infra-orbital branch, which will next be described. This nerve runs forward in several branchlets along the outer face of the articular and dentary bones to the tip of the mandible (its distal portion not shown on the plot). It supplies the skin adjacent the mandibular canal for its entire length. Its fibres were also definitely traced to three very minute naked sense organs (*m. p. l.*, Fig. 5) lying over the mandibular canal, one over the fifth canal organ, one at the fourth pore and one behind the fourth canal organ. There may also be other similar organs in the same vicinity, as they are minute and very slight imperfections in the sections might obscure them. The first one mentioned was found only on the right side of the specimen figured, though probably present on both sides.

These organs resemble in structure the similar organs lying over the opercular canal, rather than the terminal buds on the lips supplied by the r. mandibularis internus

VII. Their nerve fibres, like those of the opercular organs just referred to, are of medium or small size. That these organs are morphologically equivalent to those on the operculum, I think is clear. They therefore probably belong to the lateral line system. Whether they derive their fibres from the r. mandibularis V or from the r. mandibularis VII, I cannot determine with absolute certainty by direct observation. I think undoubtedly from the latter, for, on the one hand, the r. mandibularis V contains no other lateralis fibres, while, on the other hand, all of the similar naked opercular organs are supplied by the facialis. Moreover, it can be definitely determined that the anastomosing branch δ between the r. mandibularis V and the r. mandibularis VII contains fibres from both the V and VII nerves. Inasmuch as these fibres are all rather fine, those from the VII cannot be separately followed in Weigert preparations. In the osmic acid preparations, however, the analysis of the VII and V fibres in this nerve can easily be made, as the VII fibres, though no larger than the largest cutaneous fibres from V, yet take the metallic impregnation much more intensely. It can here be easily seen that the V fibres all, or nearly all, are given off from the recurrent nerve before it reaches the mandibularis VII, while the fibres from the latter can be separately traced cephalad after the anastomosis with the V into the branch which supplies the three mandibular organs in question. As the skin of this region was not perfectly preserved in these sections, it was impossible to trace these darker fibres into their organs. There is, in my mind, no doubt that the relations expressed in the plots are correct.

Immediately after the separation of the third branch of the r. mandibularis V, a minute twig (the 15th infra-

orbital, not drawn on the plot) separates, which passes back along one of the large tendons of the m. adductor mandibulæ. Its ultimate distribution could not be determined. It may represent a nerve for muscular or tendon sensation.

Having now reached the cephalic end of the quadrate, the r. mandibularis V now turns inward and takes its position on the inner face of the articular bone a short distance dorsad of the r. mandibularis VII. It lies immediately dorsally of Meckel's cartilage, while the mandibularis VII lies ventrally of it, and this position it maintains nearly to the tip of the mandible. Here it gives off a minute coarse-fibred twig (*io. 16*) to a separate slip of the m. adductor mandibulæ which lies mesally of the articular and dentary.

Having reached the cephalic tip of the articular bone (160), there separates from the r. mandibularis V the 17th infra-orbital, which is the second anastomosing branch for the r. mandibularis VII (*V–VII. 2*). This branch contains nearly all of the remaining coarse fibres and some of the fine and medium ones. It descends between Meckel's cartilage and the extreme tip of the articular bone to join the r. mandibularis VII and then distributes to the mm. genio hyoideus and intermandibularis and the skin of the ventral surface of the mandible and lower lip, as already described.

Farther forward the remainder of the r. mandibularis V enters a canal between the dentary bone and Meckel's cartilage and finally emerges through a foramen to the ectal aspect of the dentary bone. As it is passing through its foramen it gives off a small branch (75) and this is followed by numerous similar branches which supply the skin of the side of the mandible both cephalad and caudad

of this point and of the edge of the lower lip, to the extreme tip of the mandible.

Terminal buds are abundant near the mandibular teeth and mesally of them, but not laterally of them in the regions supplied by the fibres of this nerve, and I believe that none of these fibres are destined for these sense organs but that they are all of a general cutaneous nature. One branch, however, enters the alveolar canal of the dentary bone. Here it turns mesad and a part, if not all, of its fibres emerge again to supply the skin of the tip of the mandible near the middle line. It is probable that none of them innervate the tooth pulps, as these have a separate innervation from the r. mandibularis internus VII.

5.—The M. Adductor Mandibulæ.

This muscle is innervated in Menidia by several branches of the r. mandibularis V in the way typical for teleosts. The only exceptions to this arrangement known to me are Esox, as described by Vetter ('78, p. 496) and Lota, as described by Goronowitsch ('96, p. 41), who find that this muscle receives in addition to these fibres a small twig from the r. mandibularis VII. This needs confirmation and I may add that an attempt to trace by dissection a cutaneous nerve like my first twig of the r. mandibularis VII for the sense organ *o.6* might easily lead to such a conclusion, for the fibres pursue a tortuous and often branched course through the m. adductor mandibulæ and might easily be lost by the dissector before their emergence upon the skin.

6.—The Dorsal Division.

The dorsal division of the infra-orbital trunk contains all of the r. maxillaris V and a portion of the r. buccalis

VII, together with communis fibres from the geniculate ganglion.

After its separation from the middle division it pursues its course parallel to the latter and dorsally of it in the floor of the orbit for a considerable distance without giving off any branches.

The coarse fibres lie on the lateral side of the nerve and comprise about two-thirds of the area of its cross-section. Its first branch (the 18th infra-orbital of my enumeration, *io. 18*) is given off at 325. It contains only coarse fibres and curves around the dorsal and lateral sides of the m. adductor mandibulæ to supply the 6th organ of the infra-orbital line, and under the skin is crossed externally by a general cutaneous twig from the ventral division. The two nerves are in contact, but do not anastomose.

Immediately after giving off this branch, the dorsal division divides into two unequal portions, each containing both coarse and fine fibres. In the larger mesal portion the fine fibres are on the ventral side, though very coarse fibres are mingled among them; in the lateral portion the fine and medium fibres gather on the lateral side and very soon separate from the coarse ones (*io. 19*). These separated fine fibres distribute to the skin under the eye and about the open end of the lachrymal segment of the infra-orbital canal. The coarse fibres of the lateral portion (*io. 20*) supply the second, third, fourth and fifth canal organs of the infra-orbital line and thus belong to the r. buccalis.

The larger mesal portion of the dorsal division while in the floor of the orbit separates into mesal and lateral rami. The latter contains all of the fine fibres with a few very coarse ones scattered among them in the way so characteristic of general cutaneous nerves, and this is the

r. maxillaris (*mx. V*) in the strict sense. The former is composed wholly or nearly so of the very coarse lateralis fibres and is a portion of the r. buccalis.

The r. maxillaris, after separation from these buccalis fibres, contributes to them a general cutaneous bundle and gives off for the remainder of its coarse numerous small twigs for the skin of the side of the head in front of the eye and for the outer surface of the upper jaw. There are also given off large branches (apparently communis fibres) for the mucous lining of the jaw and the edges of the upper lip. These regions abound in taste buds and there is no doubt that these are supplied by this nerve, for there is no other obvious nerve supply. Other branches, doubtless also communis fibres, were traced into the dentary canal of the premaxillary bone and apparently innervate its teeth.

Stannius (p. 42) mentions these fibres for the mucosa of the mouth and also in several cases anastomoses with terminal twigs of the r. palatinus, which seem to be absent here. It is evidently a portion of this communis element in the r. maxillaris which corresponds to the nerve supply for the maxillary barblet of siluroids (Pollard), as suggested by Allis ('97, p. 635).

The remainder of the infra-orbital trunk, comprising lateralis and general cutaneous fibres, runs up along the inner and front walls of the orbit and then passes farther mesad and continues cephalad under the parethmoid bone along the inner side of the lower end of the olfactory fossa and under the posterior nasal aperture. It turns dorsad along the cephalic face of the lateral part of the parethmoid and laterally of the olfactory sac. While still lying close to the mucous membrane of the olfactory sac, which is not in this region sensory, it breaks up into

several branches. It is possible that some fibres pass into the olfactory mucosa, though these could not be demonstrated.

The largest of these branches supplies the first canal organ of the infra-orbital canal. Several branches pass to the skin adjacent. Three of these were definitely traced to naked sense organs lying between the anterior and posterior nasal apertures (*a. b. c.*). Finally a large branch composed mostly of coarser and deeply staining fibres passes mesad under the olfactory fossa and joins the r. ophthalmicus superficialis V. The latter nerve is at this point composed exclusively of fine general cutaneous fibres, the coarse lateralis fibres of the r. ophthalmicus superficialis VII having previously all separated from it. The coarser fibres from the r. maxillaris can, therefore, be separately followed with great ease after they have joined the ophthalmicus superficialis. They soon again withdraw and pass mesad to three large naked sense organs on the top of the snout (*d. e. f.*), one just mesally of the anterior nasal aperture, the others progressively farther cephalad and mesad, so that these three organs, together with the corresponding three of the opposite side, form nearly a perfect semicircle from one anterior nasal aperture to the other over the tip of the maxillary bone. Some of these anastomosing fibres can be traced with certainty to these three organs, but there are others which seem to end free in the skin. This anastomosing branch may carry some general cutaneous fibres.

Five of the six naked organs of the snout supplied by the r. maxillaris were found and their innervation traced on the other side of this specimen. The positions of these organs were also demonstrated in other series of sections and in surface preparations of the skin of this region. It

is not probable that there is any considerable number of superficial sense organs in this region other than those figured, for the surface preparations failed to demonstrate them, while those found were in about the same positions as figured.

The morphology of the three organs which, together with the three of the opposite side, form the supra-maxillary commissure is, I think, quite clear. They and their nerve evidently belong to the lateralis system. In Lophius (Guitel, '91) there is a commissure of the lateral lines in the corresponding position and with the same innervation (see my Fig. 6). In Amiurus and Silurus there is a similar line of pit-organs (Allis, '97, p. 629). The three organs about the nasal apertures (*a. b. c.*) and the three similar ones innervated from the superficial ophthalmic nerve (*g. h. i.*) offer much greater difficulties. The nerves supplying them are smaller than those which supply the commissural organs last mentioned, but larger and more heavily medullated than typical communis fibres so that the nerve supply here is ambiguous, as either communis or lateralis fibres might be drawn off for them. I rank them provisionally with the pit-line and other accessory lateral line organs, and suggest that the buccal group may be related to the inner buccal group of ampullæ of selachians. Comparative or embryological studies might, however, relegate them to the terminal bud system.

Cole ('98a, p. 158, foot note) describes an essentially similar nerve in Gadus from the r. buccalis to supply pit-organs in front of the nasal appertures and near the cephalic end of the supra-orbital canal. He adds: "This curious nerve is not represented in other fishes, and probably consists of lateral superficial ophthalmic fibres following a buccal course." From the quotations given

above it appears that the first supposition is not true; for this nerve seems to be general throughout the teleosts. There is no reason to assume that it is not a proper constituent of the r. buccalis.

Stannius mentions an anastomosis between the r. buccalis, in forms in which it is well isolated from the r. maxillaris (Cottus, Cyclopterus, Gadus) and the r. ophthalmicus superficialis. He has shown, furthermore ('49, p. 41) that among the teleosts there is the widest variation as to the relations of the r. mandibularis V, the r. maxillaris and the r. buccalis, from quite separate origins from the ganglionic complex to the fusion into a common infra-orbital trunk, as in the present case. As the other forms which exhibit this infra-orbital trunk belong to widely separated families, it is probable that it is merely an adaptive modification in each case. In Menidia it is clearly produced mechanically by the crowding of the parts due to the enormous size of the eyes. The fusion of the r. buccalis with the r. maxillaris he correlates in Amiurus and other forms ('49, pp. 41 and 43) with the abortion of the bones of the infra-orbital ring and this is confirmed by our relations here. On page 43 he characterizes the r. buccalis as the nerve for the region of the infra-orbital bones and includes both lateralis and general cutaneous fibres. While it is probably true in other fishes, as in Menidia, that the nerves for the organs of the infra-orbital lateral line are usually accompanied by general cutaneous fibres for the adjacent skin, yet it accords better with more recent usage to confine the term r. buccalis to the lateralis fibres and relegate the general cutaneous fibres, no matter how closely related to them, to the r. maxillaris. Thus the independence of the lateralis fibres is recognized and one step is taken toward a more consistent nomenclature.

VIII.—The Ramus Oticus.

This nerve has an independent origin from the extracranial portion of the ganglionic complex. It draws off general cutaneous and lateralis fibres, the former directly

from the Gasserian ganglion, the latter from the dorsal lateral line nerve just at the point where it divides to form the nn. buccalis and ophthalmicus superficialis VII. The r. oticus now runs cephalad and dorsad between the cranium and the m. levator arcus palatini and here divides, one twig continuing cephalad in the original position, the other directly dorsad through a foramen in the base of the post-orbital process of the frontal bone to turn caudad along the roof of the cranium under the main lateral line canal of the head. It contains 15 very coarse fibres and about 20 fine ones. Both components reach the lateral line canal through a foramen in the squamosal bone, the coarse fibres being external. These latter supply the single canal organ of the main lateral line between the opercular and the infra-orbital lines. The fine fibres could not be traced to their termini. They probably break up and lose their sheaths in the loose connective tissue surrounding the membraneous canal and possibly reach the overlying skin.

The twig of the r. oticus which is directed forward also contains both coarse and fine fibres. It turns laterally around the cephalic end of the m. levator arcus palatini and a portion of the coarse fibres enters a foramen in the most dorsal post-orbital bone to supply the single organ of the post-orbital section of the infra-orbital canal. The remaining fibres turn ventrad and anastomose with a fine-fibred nerve from the supra-orbital trunk. Fine fibres from both of these sources supply the skin around the post-orbital section of the infra-orbital canal. The coarse fibres run down under the skin close behind the eye and supply the 13th and 14th infra-orbital lateral line organs, these being the last of the series of naked infra-orbital organs. In my preliminary paper ('97) it was erroneously

stated that the n. oticus innervates two instead of three organs of the infra-orbital line.

The nerve which I have termed the otic evidently corresponds to the r. oticus + the external buccal of Gadus, as described by Cole ('98a).

Wright ('85, p. 491) holds with Van Wijhe that the r. oticus should be defined as the nerve of the neuromasts contained within the squamosal bone. In this case the term should be confined to only one of the twigs here described.

It is, I think, sound morphology to regard the r. oticus as the proper dorsal branch of the facialis segment. The sensory portion of the dorsal rami was primitively of general cutaneous nature without doubt. The morphological character of this nerve is therefore given to it by its general cutaneous rather than by its lateralis fibres and the latter accompany the former to their peripheral distribution secondarily and as a matter of mechanical convenience, just as the lateralis fibres of the r. supratemporalis vagi or glossopharyngei may (or may not) accompany the general cutaneous fibres of the dorsal ramus of the corresponding segment and just as the r. ophthalmicus superficialis VII may accompany the corresponding trigeminal nerve. This conception is justified, further, by the known relations of the r. oticus to the spiracle in forms which possess the latter structure (Wright, '85, Müller, '97, Allis, '97 and others). Pollard, ('91) finds that the r. oticus in Clarias and Auchenaspis also possess both a lateralis branch and a branch to the skin which does not go to any lateral line organ.

The r. oticus, then, was probably originally the dorsal ramus of the facial nerve to which lateralis elements have secondarily been added and whose general cutaneous portion has, like that of the profundus nerve, been cenogetically fused with the Gasserian ganglion. Compare the discussion of metamerism in Section 12.

IX.—The Truncus Supra-Orbitalis.

The supra-orbital trunk contains lateralis fibres from the dorsal lateral line ganglion, general cutaneous fibres from the Gasserian ganglion and a smaller number of communis fibres from the geniculate ganglion. The first comprise the r. ophthalmicus superficialis VII; the second the r. ophthalmicus superficialis V; the third in other fishes are usually relegated to the r. ophthalmicus superficialis V, as in Amia. The two fine-fibred components are so closely united that it is impossible to separate them far beyond the tip of the Gasserian ganglion (compare the cross-sections, Figs. 22 to 25). But the coarse-fibred lateralis component can be easily followed microscopically throughout the entire extent of the trunk. The trunk runs dorsad and cephalad along the outer face of the cranial wall under the post-orbital process and carries with it for a considerable distance an extension of the Gasserian ganglion.

Near the cephalic border of the post-orbital process, but before the tip of the ganglion has been reached, a fine-fibred branch separates dorsally (Fig. 4, *so. 1*). It seems to include both general cutaneous and communis fibres. It passes through a foramen in the sphenotic bone into the cranial cavity and then runs dorsad in the meninges. The subsequent course is closely parallel with that of the most cephalic twig of the third root of the r. lateralis accessorius, with which, however, it does not anastomose. It passes through a foramen in the frontal bone and then divides into two twigs which apparently distribute to the skin overlying the supra-orbital canal.

Slightly farther forward, *i. e.*, just at the tip of the ganglion, another fine-fibered branch separates dorsally (*so. 2*). It sends one twig laterally along the cephalic

face of the m. levator arcus palatini to anastomose with the r. oticus and supply the adjacent skin, as already described, and then it breaks up into a number of similar twigs for the adjacent skin above the eye. Of these twigs, however, one joins the r. ciliaris longus (*cil. l.*) for the dorsal side of the eye-ball and another runs out into the cornea (*co. 1*). All of these twigs are colored on the plots as if they were general cutaneous, though there is doubtless a communis element and some are probably directly derived from the sympathetic ganglion at the base of the Gasserian ganglion.

The trunk runs forward close under the lateral wing of the frontal bone and under the supra-orbital canal, the coarse fibres mostly dorsal and the fine fibres ventral, though in each case there is some admixture of fibres of the other type.

At about the level of the last (sixth) canal organ of the supra-orbital canal (430) three branches are given off—one of coarse fibres (*so. 3*) for that organ, passing through a foramen in the frontal bone to reach the canal; the second one (*so. 4*), passing mesally through a foramen in the cranial wall to the meninges of the brain at the level of the cephalic end of the optic lobes, where it turns dorsad; and the third (*so. 5*) of fine fibres, which goes cephalad parallel to and slightly dorsally of the main trunk.

The branch *so. 4* springs from the mixed ventral portion of the trunk and, like that portion, is composed of fine fibres with a few coarse ones intermingled, probably mainly, if not wholly, general cutaneous. It passes dorsad in the meninges and divides into numerous very fine branches which anastomose more or less with each other, but apparently not with any of the other meningeal rami. Two of

these branches pierce the frontal bone by separate foramina and distribute to the skin of the top of the head over the supra-orbital canal. No sense organs were found in this vicinity. Others pass to the dorsal side of the brain and there in its membranes unite into an intricate plexus which envelops the large pineal vesicle which lies under the skull and over the extreme cephalic tip of the optic lobes and the caudal part of the cerebrum. Some fibres of this plexus pass to the median line cephalad of the epiphysis and there in the meninges unite with a similar fascicle from the other side. The nerve thus formed runs cephalad exactly in the median line between the brain membranes and the exceedingly delicate pallium to the tip of the cerebrum. It then passes dorsad through the cranial cavity to the inner side of the cranial roof in which position it continues forward immediately under the suture of the two frontal bones. Still farther cephalad it lies in the narrow space between the frontal bones and the internasal cartilage, nearly to the tip of the latter. It could be followed beyond the cephalic end of the frontal bones, in the latter part of its course leaving the dorsal surface of the internasal cartilage to run in the subcutaneous connective tissue, where it is finally lost.

This peculiar nerve apparently corresponds to the "intra-cranial ascending dorsal twigs of the N. trigeminus and N. facialis," which in Silurus arise from the ganglionic complex cephalad of the r. lateralis V and run forward intra-cranially and under the skin of the head, one branch ramifying over the nasal bone (Stannius, '49, p. 48). Whether it is of sympathetic, communis or general cutaneous nature, I have no means of deciding positively. As indicated on the plots, I believe that it belongs mainly to the latter component. This, however, needs confirmation.

The branch *so.* 5 runs close to the trunk, but separated from it by a large blood vessel, and gives off numerous branches for the skin between the eye and the supra-orbital canal. As it approaches the cephalic edge of the eye, it diverges laterally from the trunk and sends several twigs to the cornea (*co.* 2) and eye-ball. The remaining fibres supply a thickened fold of skin laterally of the nasal openings. None were traced to special sense organs, the buds in the cephalic part of this region having a different nerve supply.

At the level of the fifth canal organ of the supra-orbital line a coarse-fibred branch (*so.* 6) leaves the trunk to supply this and the fourth organ, these two organs lying very close together, but their nerves passing into the canal through separate foramina in the frontal bone.

Under the pore between the third and the fourth organs of the supra-orbital canal a branch (*so.* 7), comprising fine and medium fibres, passes up through a foramen in the bony floor of the canal to the skin of the top of the head, part of the fibres running forward a long distance within the bony canal, finally to emerge to the overlying skin.

Then follow two coarse-fibred branches (*so.* 8 and *so.* 9) for the third and second supra-orbital canal organs, each with its special foramen in the frontal bone.

The trunk meanwhile runs parallel with and close under the canal, being separated from it only by the frontal bone. This bone consists of two broad wings and a short vertical plate, the canal lying at the point of their intersection. One of the wings runs inward from the canal, over the brain cavity and internasal cartilage, the other outward over the eye, while the vertical plate runs down from the canal along the lateral face of the supra-orbital and internasal cartilages. The supra-orbital trunk runs

in the angle between the vertical plate and the lateral wing, parallel with the slender supra-orbital cartilage and the dorso-lateral edge of the massive internasal cartilage. At the point where the branch *so. 9* for the second canal organ is given off the internasal cartilage spreads out laterally under the frontal bone, so that the supra-orbital trunk lies in a canal bounded above by the frontal bone and on all other sides by the cartilage. This lateral projection of the internasal cartilage is covered on its cephalic and lateral aspects by the highly developed par-ethmoid ossification and both the cartilage and its investing bone imperfectly enclose the caudal part of the olfactory sac. From that portion of the par-ethmoid which lies behind the olfactory fossa a V-shaped tongue of bone extends into the substance of the internasal cartilage to form a partial bony wall to the canal which contains the olfactory nerve, and a similar tongue farther dorsad to form the floor of the canal containing the supra-orbital trunk, so that this trunk for some distance before it emerges into the olfactory fossa lies in a deep canal bounded above by the frontal and below by the par-ethmoid.

It emerges at about the same transverse level as the olfactory nerve, but by a separate foramen farther dorsally, previously, however, giving off a very slender nerve (*so. 10*), which turns ventrad and passes into the olfactory fossa by a separate foramen in the par-ethmoid bone and then runs cephalad along the lateral face of the olfactory sac, where it finally joins one of the dorsal twigs of the r. maxillaris V. It probably supplies the caudal (non-sensory) portions of the walls of the olfactory sac.

Immediately upon the emergence of the trunk from its canal a fine-fibred branch (*so. 11*) separates and goes at once to the skin mesally of the post-nasal aperture. The

trunk now continues forward in its former position ventrally of the supra-orbital canal and separated from it by the nasal bone. Mesally of it is the internasal cartilage, laterally the nasal sac and ventrally the olfactory nerve.

Under the caudal end of the nasal bone several twigs are given off, one of fine fibres mesally for the skin (not figured), one laterally of fine and medium fibres (*so. 12*), and two dorsally (*so. 13* and *so. 14*), each of coarse and medium fibres, leaving in the main trunk only very fine fibres with a few coarse ones scattered among them, the typical general cutaneous arrangement. One of the dorsal twigs (*so. 13*) supplies the first canal organ; the other (*so. 14*) and the lateral twig (*so. 12*) turn laterad along the dorsal wall of the nasal sac, anastomose with each other, and terminate in three large naked sense organs (*g*, *h*, *i*) between the dorsal margin of the anterior nasal aperture and the cephalic end of the supra-orbital canal. This arrangement was confirmed on the opposite side of this specimen and the three organs were seen in other sections and in surface preparations of the skin of this region.

From the trunk other cutaneous twigs directed inward go off from time to time and under the first canal organ it receives the anastomosing branch from the infra-orbital trunk. The latter passes between two separated bundles of fibres of the supra-orbital trunk to the naked sense organs *d*, *e*, *f*, above the maxillary bone, as already described. It is possible that some fibres from the infra-orbital trunk remain in the supra-orbital trunk, but certainly no considerable number do so.

After the anastomosis the fibres of the trunk distribute to the skin of the top of the snout to the extreme tip of the upper lip. Near the end of the premaxillary bone a

large branch enters the dentary canal of that bone and turns back in it, probably supplying the teeth. These are, doubtless, communis fibres.

The three sense organs, *g*, *h*, *i*, above the anterior nasal aperture which are supplied by the superficial ophthalmic nerve resemble those of the same neighborhood which are supplied by the infra-orbital trunk and they are, doubtless, of the same nature. The same ambiguity holds here as there; I incline to the belief that they correspond to pit-organs.

Communis fibres can be clearly traced into the supra-orbital trunk. What their distribution may be is not so clear. Apparently they are of a simple visceral nature for the internal organs of the head. Terminal buds are known to be supplied by communis fibres of the r. ophthalmicus superficialis in some fishes (*e. g.*, Amia, Allis, '97). The naked sense organs on the barblets of siluroids are unquestionably supplied by communis fibres. My own preparations of Amiurus substantiate this. The nasal barblet is stated by Wright ('84, p. 367) to be innervated by the r. ophthalmicus profundus in Amiurus; this nerve is, however, the ophthalmicus superficialis, as Allis has suggested ('97, p. 539).

Stannius clearly recognized the lateralis and general cutaneous components of this supra-orbital trunk and the origin and distribution of each. The lateralis component he identified with the r. frontalis, the general cutaneous with the r. nasalis of higher vertebrates (p. 35), the former of course being an impossible homology.

Goronowitsch describes the nerves entering this trunk in Lota vulgaris but erroneously names the ventral, or trigeminal, nerve ('96, p. 27) the r. ophthalmicus profundus. From his brief statement of the peripheral distribution this is clearly impossible. He describes and figures in this nerve (*i. e.*, the r. ophthalmicus superficialis V) a large bundle of communis fibres from the communis root of the facial (his dorsal root of the VII nerve).

X.—The R. Lateralis Accessorius.

This nerve receives communis fibres from the vagus and from the facialis. The latter arise from the geniculate ganglion by several small roots, which will be enumerated from behind forward. The first and largest of these recurrent roots (*rec. 1*) arises from the most caudal portion of the geniculate ganglion in several strands. Their relations are indicated somewhat diagrammatically in Fig. 26, which is a composite of several successive camera outlines of this region. Three of them pass directly dorsad over the emerging spinal V root and mesally of the dorsal lateral line root, while one arises farther forward than the others from the ventral surface of the ganglion and passes dorsad and caudad mesally of the sensory V root just after its emergence from the oblongata and of all the other V + VII roots, but laterally of the IV root. This root it follows back to its origin and then joins the other strands on the dorsal side of the dorsal lateral line root. This strand has a double origin, the two portions separately entering the IV nerve, which they closely follow and from which they separate together. The root as thus composed then continues dorsad into the meninges at the level of the caudal end of the optic lobe, then through a foramen in the cranial roof to turn caudad under the skin. It is composed chiefly of very fine fibres with a few more densely myelinated fibres of medium size scattered among them.

From the cephalic tip of the geniculate ganglion, after the separation of the fibres which go out through the hyomandibular foramen, the remaining communis fibres divide into two bundles. The larger one goes out ventrally with the infra-orbital trunk, as already described; the smaller one curves around the outer side of the

sensory V root dorsally. Just caudad of the point where the fibres of the dorsal lateral line root diverge toward their respective trunks this dorsal bundle of communis fibres sends off the second recurrent root (*rec. 2*). This root is then re-enforced by a strand of communis fibres from the ventral bundle. It goes directly dorsad into the meninges covering the optic lobe. Here it breaks up into several small twigs, the largest of which continues caudad to join the first root before it leaves the cranium. Another twig runs in the meninges farther dorsad, where it is joined by a small twig from the third root of the r. lateralis accessorious, and after the anastomosis it at once sends a twig dorsad through a foramen in the cranial roof. The terminus of this twig was lost by imperfections in the sections. I find, however, on the opposite side of the same specimen a naked sense organ in the corresponding position, which is doubtless supplied by this nerve. The remaining fibres of this anastomosing twig run to a foramen in the roof of the cranium farther caudad and probably join the first recurrent root extra-cranially.

The third root of the r. lateralis accessorius (*rec. 3*) arises immediately cephalad of the second and close behind the origin of the r. oticus. It contains very fine fibres and a considerable number of slightly larger and more heavily myelinated fibres. The latter arise from the dorsal communis bundle. Near its origin and embedded among its fibres is a cluster of five or six small ganglion cells. They belong to the finer fibres, and these enter the V + VII ganglionic complex farther cephalad than the other fibres. In several of my series these finest fibres were traced cephalad with tolerable certainty into the most cephalic ganglion (*sy. 1*) of the sympathetic chain, as indicated on Fig. 4. This little ganglion is,

therefore, almost certainly sympathetic. This root, like the preceding, runs dorsad in the meninges and then breaks up into several minute twigs. The largest of these runs back and within the cranium joins the most cephalic branch of the second root. Of the other twigs some appear to supply the meninges, but most, and these containing the coarser fibres, rise to the cranial roof, which they perforate, each by a minute foramen in the frontal bone. Five such branches were followed and of these three could be traced to naked sense organs on the dorsal surface of the head. Probably the others have similar destinations.

On the opposite side of the specimen plotted the details of the facial roots of the r. lateralis accessorius are somewhat different. The first root arises by several strands essentially as figured for the left side, though the details of their arrangement are not exactly the same. The second root is wanting altogether, and as this is the case on both sides of another specimen examined, I assume it to be the more usual arrangement. The third root is about as figured, though not exactly. As before, it consists of some very fine fibres and some a little coarser with heavier myelination. The latter come from behind and clearly from the communis, the former arise a little farther cephalad and probably from the sympathetic and are provided with the little ganglion. The third root breaks up into numerous branches, some of which were traced to sense organs, as on the other side. In the upper part of the cranial cavity this root sends back a large branch which joins the main r. lateralis accessorius from the first root.

As the large scales covering the top of the head make it difficult to get perfect sections, several surface preparations of the skin of this region were made to control the sections. There are undoubtedly some more organs in the region overlying the optic lobes than the plots indicate, but that number is not large and is probably not

greater than the number of nerves which penetrate the cranium. The dorsal surface does not present superficial organs in regions other than this one.

The three meningeal nerves just described as roots of the r. lateralis accessorius all have intra-cranial origins and pursue essentially similar courses. They are, moreover, all bound together in an intricate and more or less variable plexus. There are farther cephalad two meningeal nerves which have extra-cranial origins from the supra-orbital trunk and which do not enter this plexus but are destined chiefly, at least, for the skin of the top of the head farther cephalad. I regard them as primarily general cutaneous nerves and as such have described them in the preceding pages. They are, however, doubtless accompanied by sympathetic or other visceral fibres and they may participate somewhat in the general meningeal plexus. Indeed the whole plexus is vastly more complicated than my diagrams indicate, and only the larger nerves could be traced with precision.

These organs overlying the optic lobes, which I have assumed to be terminal buds, are structurally similar to those about the nasal apertures, which I regard as pit-organs. The nerve supply here also is not absolutely free from ambiguity, and I must admit the possibility that these fibres are derived from the lateralis roots by an intra-cranial anastomosis which I have overlooked.

Almost directly dorsad of the origin of the first root of the r. lateralis accessorius and after receiving fibres from the other roots, which vary in number and importance in different individuals, the main r. lateralis accessorius turns abruptly caudad, first, however, receiving the anastomosing fibres from the most cephalic twig of the r. supra-temporalis vagi, as described under that nerve. It runs closely appressed to the outer surface of the cranium not far from the median line and contains very small fibres with a few of slightly larger calibre which are much

more heavily myelinated. It gives off a few fibres from time to time which join the rich sub-cutaneous plexus in which they could not be further traced.

When the dorsal musculature begins to appear the r. lateralis accessorius follows the dorsal surface of this muscle close under the skin and at about this level (600) it receives the second anastomosing branch from the r. supra-temporalis vagi. A short distance farther caudad it receives two anastomosing branches from the dorsal r. communicans *b* of the first spinal nerve, which break through the dorsal musculature in the septum between the general dorsal musculature and the interspinal muscles, nearer the median line. The r. lateralis accessorius now sinks down a short distance into the same intermuscular septum, in which it continues into the trunk. It receives a third anastomosing branch from the first spinal and from this point caudad one such branch for each segment. The first of these is formed by the union of one nerve from the first spinal nerve and one from the second, the second by one from the second spinal and one from the third, and so on. See the account of the spinal nerves, Section 4. Just caudad of the level of the third spinal ganglion the r. lateralis accessorius is joined by the third anastomosing branch from the r. lateralis vagi.

The discussion of the morphology of the r. lateralis accessorius is deferred until Section 12, which see.

XI.—The Ramus Ophthalmicus Profundus.

This nerve is said by most other authors to be absent in the bony fishes, except in the siluroids. I find, however, that a portion of the Gasserian ganglion has been isolated from the rest and fused more or less closely with the most cephalic ganglion of the sympathetic chain and

that from these ganglion cells a nerve is given off which accompanies the radix ciliaris longa to the ciliary ganglion (see Fig. 4). These general cutaneous fibres I tentatively homologize with the r. ophthalmicus profundus. They can best be described in connection with the account of the sympathetic nerves which they accompany; their detailed description and the figures illustrating them will, accordingly, be given in the next section.

The character of these trigeminal fibres in the radix longa of the ciliary ganglion is a matter of great theoretical interest. Van Wijhe, Beard and many others (Marshall and Spencer, '81; Ewart, '89 and '93; Platt, '91; Neal, '98, etc.), as is well known, give in elasmobranchs to the r. ophthalmicus profundus or its embryonic precursor the rank of a separate sensory nerve whose motor part may be represented in the oculomotorius. Or, according to other authors, the motor root of the profundus has disappeared in higher vertebrates, being represented as such in myxinoids.

Its ganglion ("g. mesocephali") often has only a temporary separate existence and fuses with the Gasserian ganglion in the adult. The suggestion (Schwalbe, '79, and others) that the ciliary ganglion is the vestige of the mesocephalic ganglion is apparently discredited by the accumulating evidence that the former ganglion is composed of sympathetic cells only (Retzius, '94 and '94a; Michel, '94; v. Kölliker, '94; Huber, '97).

The profundus nerve of siluroids, as described by Wright ('84) in Amiurus and by Pollard in Clarias and Trichomycterus ('95) requires further study. Allis thinks in the latter case that it is the r. ophthalmicus superficialis V and not the profundus, and the same seems to be the case in Amiurus also.

Trigla is the only one of the teleosts for which anything like the condition in Menidia has been described. Stannius (p. 25) mentions a fine-fibred nerve which separates from the trigeminus root intra-cranially and after emerging by a separate foramen into the orbit enters a small ganglion. From this ganglion, which is, undoubtedly, the profundus ganglion, as Allis ('97, p. 538) has maintained, there are given off a ramus ciliaris longus and a radix longa ad ganglion ciliare, very much as in Menidia.

Allis in the passage last cited has given a very comprehensive review and critique of the literature of the ophthalmicus profundus which need not be again summarized here. The primitive profundus nerve probably contained dorsal and ventral branches. The former is represented by the portio ophthalmici profundi of Amia and in teleosts it is either fused with, or supplanted by, the r. ophthalmicus superficialis trigemini. It cannot be identical with the latter nerve for in several forms both nerves are present. With it may be associated more or less closely the ramus ciliaris longus. The ventral branch comprises the ophthalmicus profundus of selachians and most higher forms and is associated with the radix longa of the ciliary ganglion.

Amia, as usual, exhibits a transitional stage in the evolution of the teleostean specialization. Here the profundus root has fused with the V root, but the ganglion is widely separated. From the dorsal angle of the ganglion is given off the large portio ophthalmici profundi, which joins the r. ophthalmicus superficialis V. The two rami ciliares longi arise in connection with it from the profundus ganglion. From the ventral angle of the ganglion the radix longa is given off, while the very small r. ophthalmicus profundus *sensu stricto* arises from the gan-

glion between the dorsal and ventral angles. This latter nerve is in a very interesting condition, and it would appear from Allis' description to be in a state of degeneration (p. 533)—"When this last nerve was found, it always accompanied the ciliary nerves as they ran forward and outward between the external and superior recti. Beyond that point it was always lost, appearing sometimes to fuse with the ciliary nerves, and at others to disappear in the general tissues."

In Menidia the profundus is still further reduced and more intimately fused with the trigeminus. The portio ophthalmici profundi is lost and the r. ophthalmicus profundus fused for its entire length with the radix longa.

The peculiar and constant relations of the ophthalmicus to the sympathetic are not difficut of explanation. The "head part" of the sympathetic has one or more ganglia associated with the ganglia of all of the cranial nerves, doubtless including the primitive profundus. Now, the sympathetic ganglion lying under the primitive profundus glanglion having become connected with the oculomotor nerve (either secondarily or primarily, if the III nerve should prove to be the motor nerve of the profundus segment, as some maintain), it was retained in this position during the backward migration of the profundus ganglion toward the Gasserian, and now appears as the ciliary ganglion.

Allis remarks that the superficialis trigemini and the profundus seem to vary in relative importance directly as the number of terminal buds found on the top of the head and snout. The primary composition of these nerves is, it seems to me, undoubtedly general cutaneous rather than special cutaneous for terminal buds. The number of the latter fibres is certainly an important factor, never-

theless it must not be forgotten that independently of that the size of these nerves will be determined largely by the relative development of the different parts of the head. Thus in the selachians the development of the rostrum, which is undoubtedly a dorsal region, has necessitated a large increase in the general cutaneous nerve supply. In such teleosts as Menidia, on the other hand, the dorsal surface of the head over and in front of the eyes has been reduced to a minimum, with a corresponding loss in the nerve supply.

XII.—Comparison with Acipenser and Lota.

We have in our discussion of the nerve roots earlier in this section called attention to the failure of Goronowitsch's segmental scheme of the trigemino-facial nerves ('88 and '96, especially the latter). He arranges, it will be recalled, the trigemino-facial roots in three homodynamous series, each with dorsal sensory and ventral motor roots, (1) trigeminus I (my general cutaneous and motor V roots), (2) trigeminus II (my two lateralis roots), (3) facial (my communis and motor VII). In this very attractive scheme there are two fatal defects. The first is that the ventral root of his trigeminus II is sensory, not motor. The second and more radical difficulty arises out of the fact that Goronowitsch considers disparate structures to be serially homologous. Thus, of the three dorsal roots in question which he considers to be homologous with each other and with the spinal dorsal roots, the first is general cutaneous, the second is lateralis and the third is communis. Now, it is of course possible that future researches may show that this root complex represents two or three or more primary metameres; but the origin and distribution of these root fibres in the adult certainly negative any such

direct comparison as that which Goronowitsch attempts to draw.

Goronowitsch's homologies of the rami in the case of Lota are also in some cases confusing. The truncus hyomandibularis is, as we have seen, composed exactly as in Menidia, save that the general cutaneous component is larger. This component he states is absent altogether in Acipenser, *i. e.*, there is no anastomosis from the trigeminus I to the hyomandibularis. The ophthalmicus superficialis VII in Lota is as in Menidia. The ophthalmicus superficialis V, which he incorrectly calls the ophthalmicus profundus, has in addition to general cutaneous fibres from trigeminus I, a large bundle from his facialis, which must be of communis nature, and may supply terminal buds of the top of the head as Allis describes in Amia.

Regarding the maxillary and mandibular nerves there is considerable confusion, which, however, can be cleared up, I think, by comparisons with Menidia. The composition of these nerves in Acipenser is probably as follows: Goronowitsch describes a rostral nerve from each of the three nerves, trigeminus I, trigeminus II and facialis. The first of these is from its origin evidently the proper r. maxillaris (general cutaneous) and it is described as innervating the appropriate cutaneous area. The second is equally clearly the r. buccalis and it accordingly supplies the infra-orbital canal organs. The third arises from the facial (geniculate) ganglion and is accordingly a communis nerve. This is the n. rostri interni of Stannius. It distributes to the upper lip and particularly to the barbels. It is, I think, the homologue of the communis fibres contained in the r. maxillaris of Menidia and distributed to the taste buds of the upper lip. In Acipenser there is anastomosis of these three nerves

peripherally and some confusion of the relations, but I predict that microscopical examination will reveal essentially the arrangement which I have given and that the nerves will not prove to be serially homologous structures as Goronowitsch assumes. In Acipenser there is also a r. palatinus which comes from the facial and is doubtless purely communis, as usual. The r. mandibularis V is derived wholly from trigeminus I. Goronowitsch regards it apparently as a pure motor nerve ('88, p. 479), and that, too, in spite of the fact that he found ganglion cells running out into its trunk. It doubtless contains general cutaneous fibres also.

In Lota, Esox and Gobio the first two rostral nerves were found (viz., my maxillary—the general cutaneous portion—and my buccal), but the facial (communis) rostral nerve was not found. Lota has a palatine nerve which is strictly typical. There is in addition a large bundle of facial (communis) fibres which joins itself to general cutaneous fibres from the trigeminus I and enters the r. mandibularis V. Now, Goronowitsch, impressed with the necessity of finding a homologue in Lota of the rostral nerve of his third segment in Acipenser, identifies the r. palatinus of Lota with the r. rostri interni of Acipenser and then assumes that the facialis fibres which enter the r. mandibularis V in Lota correspond to the r. palatinus of Acipenser. These homologies seem impossible, for the distribution area of the r. palatinus is not at all that of the r. rostri interni, and how can a lower jaw nerve be homologous with an upper jaw nerve? Allis ('97) finds communis fibres entering the r. mandibularis V in Amia and these distribute to terminal buds of the outer skin and mucous surfaces in the mouth, both of the hyoid region, and not at all to the palatine region, and it is

probable that these correspond to the facialis fibres in the corresponding nerve of Lota. The palatine nerve of Lota and other teleosts is unquestionably the same as the nerve of that name in ganoids and other forms. The communis fibres for the upper lip which correspond to the r. rostri interni of Acipenser, if present in Lota, doubtless go out with the r. maxillaris as in Menidia, and were overlooked by Goronowitsch. I have personal knowledge that this is the case in Gadus.

XIII.—Summary of the Trigemino-Facial Complex.

The trigemino-facial roots and ganglia can be clearly separated and the peripheral distribution of each has been traced.

The motor VII supplies muscles belonging to the constrictor system of the facialis segment. It does not supply the geniohyoideus and intermandibularis muscles, these being supplied by the motor V. The r. hyoideus is a mixed nerve, motor and general cutaneous; the r. mandibularis VII is purely sensory, though two components are represented, corresponding to the r. mandibularis externus (lateralis) and the r. mandibularis internus (communis) of the Amphibia. It contains no general cutaneous fibres save those derived by peripheral anastomosis from the r. mandibularis V.

There is a pre-trematic branch of the facial nerve, which has an independent course in Menidia and innervates the large (spiracular) pseudobranch and the mucosa adjacent. This is not the chorda tympani of higher forms and I consider it probable that this nerve is absent in Menidia, though present in some other fishes.

The accompanying tables express the relations of the roots, ganglia and rami.

A.—Facialis Complex.

Roots.	Central Termini.	Ganglia.	Rami.
I.—Communis ("dorsal geniculate")	fasciculus communis	geniculate	1. truncus hyomandibularis—r. man. int. VII. 2. r. pre-trematicus VII. 3. r. palatinus VII. 4. truncus infra-orbitalis—internal portion of r. maxillaris V. 5. truncus supra-orbitalis—in part. 6. facial root of r. lateralis accessorius.
II.—Motor	motor VII nuc. and fasc. long. dors.?	————	truncus hyomandibularis (a) r. opercularis profundus. (b) r. hyoideus—motor component.
III.—Ventral lateralis root	tuberculum acusticum	ventral lateral g.	truncus hyomandibularis (a) r. opercularis superficialis—lateralis component. (b) r. mandibularis externus VII.
IV.—Dorsal lateralis root	tuberculum acusticum	dorsal lateral g.	1. truncus infra-orbitalis—r. buccalis. 2. truncus supra-orbitalis—r. ophth. sup. VII. 3. r. oticus—lateralis component.

B.—Trigeminus Complex.

Roots.	Central Termini.	Ganglia.	Rami.
I.—General cutaneous	spinal V tract and chief V nucleus	Gasserian	1. truncus hyomandibularis (a) r. operc. superficialis—gen. cut. comp. (b) r. hyoideus—gen. cut. comp. 2. truncus infra-orbitalis (a) r. mandibularis V—gen. cut. comp. (b) r. maxillaris V—gen. cut. comp. 3. truncus supra-orbitalis—r. ophth. sup. V. 4. r. oticus—gen. cut. comp. 5. r. ophthalmicus profundus.
II.—Motor	motor V nuc. and fasc. long. dors.?	————	truncus infra-orbitalis—r. mandib. V, motor component.

The infra-orbital trunk is a fusion of four nerves which in some vertebrates are distinct. These are, (1) the r. mandibularis V, with general cutaneous and motor components, (2) the r. maxillaris V, general cutaneous, (3) the r. buccalis, lateralis, and (4) communis fibres to taste buds of the upper lip, which apparently correspond to the n. rostri interni (Stannius) of Acipenser.

The supra-orbital trunk is a fusion of the r. ophthalmicus superficialis VII, for the supra-orbital lateral line, communis fibres of uncertain distribution, and the r. ophthalmicus superficialis V, general cutaneous. The latter nerve must not be confused with the r. ophthalmicus profundus, as several of the most recent writers have done. The profundus nerve is apparently represented by a vestigeal bundle of general cutaneous fibres which run out from the Gasserian ganglion with the radix longa of the ciliary ganglion.

The r. oticus is the dorsal ramus of the facialis segment and was probably originally a general cutaneous nerve to which a lateralis element has been added. Its general cutaneous portion has secondarily fused with the Gasserian ganglion.

The r. lateralis accessorius corresponds to the r. recurrens V, or superficial lateral line nerve, of the older authors. It is composed exclusively of communis fibres and, after receiving other communis roots from the vagus complex, seems to innervate the row of terminal buds under the dorsal fin.

Section 8.—The Sympathetic Nervous System.

The sympathetic system has not been exhaustively studied, as the work of previous investigators has covered the ground quite satisfactorily so far as it can be done

with the methods here employed. The topographical relations of the ganglia and the larger sympathetic nerves are indicated upon Fig. 4, and will be here briefly reviewed, though they deviate but little from Belone and other well-known examples, for which, compare the descriptions of Stannius.

My examination began on the left side at the level of the fourth vertebra, where the sympathetic trunk of each side lies close to the centrum, and the description will proceed cephalad from this point. The fibres are small but quite heavily myelinated. They are considerably larger than the sympathetic fibres which go out with the cranial nerves. Immediately caudad of the fourth spinal ganglion, cells appear in the sympathetic cord. This sympathetic ganglion is very small and ceases before the spinal ganglion is reached. Now, instead of sending a r. communicans to the ventral ramus of the spinal nerve in the usual manner, the whole sympathetic trunk rises up and becomes embedded in the ventral side of the spinal ganglion and here additional sympathetic ganglion cells are found. They can be distinguished from the cells of the spinal ganglion by their smaller size. Sympathetic fibres and a portion of the sympathetic ganglion run out into the ventral ramus. A bundle of fibres passes also from the sympathetic ganglion across the root of the ventral ramus and back into the spinal ganglion.

The sympathetic ganglion runs somewhat farther cephalad than the spinal ganglion and from its tip the sympathetic trunk again descends to its former position, laterally of the centrum of the vertebra, in this case, however, separated from the latter by the fleshy origin of the m. retractor arcus branchii dorsalis, so that it lies between this muscle and the head kidney close to the dorso-mesal angle of the latter.

Between the fourth and the third spinal ganglia no ganglion cells appear in the sympathetic trunk, which, however, rises up and again becomes embedded in the third spinal ganglion, where it develops a ganglion of its own, as in the previous case. Some of the sympathetic fibres pass through the ganglion without losing their medullary sheaths. This sympathetic ganglion sends fibres from its cephalic end into the ventral ramus and others, as before, back into the spinal ganglion.

The sympathetic trunk now resumes its former position between the m. retractor arcus branchii dorsalis and the head kidney nearly up to the level of the second spinal ganglion. Here a sympathetic ganglion is found, which fuses with the caudal end of the spinal ganglion and the sympathetic trunk runs embedded in the latter to its cephalic tip, where there is another sympathetic ganglion, which effects the usual relations with the ventral ramus (*r. v. 2*). The trunk then again passes down to its former position.

Opposite the second vertebra there is another ganglionic enlargement which extends forward as far as the r. ventralis of the first spinal nerve (*r. v. 1*). The nerve trunk, however, turns ventrad just cephalad of the m. retractor arcus branchii dorsalis and enters a large ganglion (*sy. 7*), within which it divides, one part continuing cephalad, the other passing ventrad and mesad to a small ganglion cœliacum (*g. cœl.*) lying ventrally and laterally of the centrum of the second vertebra. From this ganglion a strong sympathetic commissural nerve runs under the vertebra to a similar ganglion of the opposite (right) side, which in turn is in like manner related to a ganglion of the chain of that side and from which the n. splanchnicus is given off. The latter nerve is confined to the right

side. Of the various modes of origin of this nerve among the bony fishes enumerated by Stannius (p. 138) the arrangement here conforms most closely to that of Belone. The commissural nerve as figured (*sy. c.*) is the left root of the splanchnic nerve.

The "head part" of the sympathetic trunk may be regarded as beginning from the same ganglion (*sy. 7*), which gives rise to the root of the cœliac ganglion (Stannius' usage). It runs cephalad, as before, along the dorsal surface of the head kidney, and under the ganglion of the first spinal nerve there is found the last ganglion of the "head part," the sixth from in front (*sy. 6*), not counting the ciliary ganglion. This ganglion is much larger than any of the preceding. It lies close under the ganglion of the r. lateralis vagi and may send some fibres into it. In front it fuses broadly with the caudal part of the vagus ganglion, being particularly intimately united with that portion which I have termed the jugular ganglion. As in the preceding cases, some of the fibres pass through the ganglion without losing their medullary sheaths.

The sympathetic trunk now separates from the vagus ganglionic complex slightly and runs along its inner and ventral aspect between it and the lower outer edge of the vagus foramen. Just cephalad of the closure of that foramen ganglionic cells reappear in the trunk. This small ganglion (*sy. 5*) lies between the vagus ganglion and the emerging root of the glossopharyngeus, but does not communicate with either. The trunk now fuses with the IX root in which it can be separately followed to the IX ganglion. Just before reaching the latter it withdraws from the IX root, but follows along its dorsal surface as a separate round bundle and here there is another minute sympathetic ganglion (*sy. 4*).

Separating from the IX ganglion, the trunk follows a big blood vessel along the outer wall of the cranium nearly to the foramen of the truncus hyomandibularis, running at the level of the ventral edge of the membranous labyrinth, which lies within the cranium. Throughout this portion of its course ganglion cells are scattered freely along the trunk, which has about the same number and character of fibres as in its post-vagal course through the body. Along with the vessel above referred to, it enters a foramen in the cranial wall which communicates with that of the truncus hyomandibularis, and a sympathetic ganglion which is here formed (*sy. 3*) applies itself closely to the ventral side of the mixed truncus hyomandibularis and r. palatinus within this foramen, and a portion runs out into these nerves. The ganglionic chain continues (*sy. 2*) into a similar foramen cephalad of that of the truncus hyomandibularis, which is occupied by a large blood sinus and by the caudal extension of the Gasserian ganglion from which arises the caudal root of the general cutaneous component of the truncus hyomandibularis, where it applies itself to the ventral side of the extra-cranial part of the Gasserian ganglion (Fig. 2, *sy. 2*) and follows it back into the cranium.

From this point cephalad the sympathetic ganglion is intimately fused with the ventral face of the Gasserian ganglion and sympathetic fibres can be seen to enter that ganglion. Others were traced into the infra-orbital and supra-orbital trunks and, as before stated, into the r. lateralis accessorius.

Sanders ('79, p. 745) describes in Merlangus an extra-cranial connection between the IX ganglion and the Gasserian, which conforms to my sympathetic nerve. He, however, does not regard it as sympathetic, but errone-

ously identifies the latter with the intra-cranial r. recurrens of the carp.

Cole ('98a, p. 145) has shown that in many fishes the r. pharyngeus IX may run forward accompanying this sympathetic nerve to join the r. palatinus VII, thus constituting a true Jacobson's anastomosis like that of the mammals. In Menidia the absense of the r. pre-trematicus IX is correlated with the reduction of the r. pharyngeus IX. The sympathetic ganglion *sy. 4* has, however, a slight fibrous connection with the IX ganglion and among these fibres there may be a few of the proper glossopharyngeal fibres which run forward with the sympathetic as a Jacobson's anastomosis. If such fibres occur, they cannot be distinguished from the sympathetic fibres and in any case their number would be very small. The true nature of this anastomosis, where it occurs, was recognized by Stannius ('49, p. 58, foot-note 1), though he did not make the homology with the Jacobson's anastomosis.

Continuing cephalad from the first sympathetic ganglion on the ventral edge of the Gasserian ganglion are two sympathetic nerves, one the radix longa of the ciliary ganglion, the other farther dorsad, the ramus ciliaris longus, which pursues an independent course to the eyeball. The sympathetic ganglion from which they spring (*sy. 1*) is very intimately related to the Gasserian and in places can be distinguished from it only by the small size of its cells, though farther caudad the two ganglia are quite distinct (Fig. 23). It then becomes bi-lobed (Fig. 24), the median lobe having only the characteristic small sympathetic cells and giving off a branch (*sy. rec. 3*) dorsad through the substance of the Gasserian ganglion for the third root of the r. lateralis accessorius, and farther

cephalad (*sy. oph. sup.*, Fig. 25) running into the sympathetic strand for the supra-orbital trunk, finally to terminate in the ramus ciliaris longus. The more lateral lobe receives a strong bundle of medullated root fibr s from the Gasserian ganglion (*o. pr. r.* Fig. 24) and contains mingled among the sympathetic ganglion cells larger ones which apparently belong to the fibres from the trigeminus root and which become more numerous farther cephalad. The radix longa of the ciliary ganglion arises from this lateral lobe (Fig. 25) and apparently contains both sympathetic and trigeminal fibres. All of the fibres from the lateral lobe enter the radix ciliaris longa.

These fibres from the Gasserian ganglion to the radix longa are presumably of general cutaneous and not sympathetic nature. They could not be separately followed through the ciliary ganglion and hence their peripheral distribution is unknown. Their morphology is discussed in Section 7, XI.

From the lateral lobe of the first sympathetic ganglion a minute sympathetic twig goes off ventrad and joins the ventral ramus of the n. oculomotorius just after the separation of the dorsal ramus and just before its anastomosis (radix brevis) with the ciliary ganglion.

The ramus ciliaris brevis arises from the ciliary ganglion, which is provided with two roots in the typical way, the radix longa from the Gasserian ganglion and the radix brevis from the III nerve. The radix longa is composed mostly of small and medium-sized fibres with a few very large ones. It turns abruptly mesad and ventrad to the ciliary ganglion which lies in contact with the III nerve.

The ciliary ganglion is composed for the most part of the very small cells so characteristic of all of the ganglia

of the sympathetic system, but among them are some of larger size like those of the Gasserian ganglion. The caudal end of the ganglion is composed chiefly of the larger cells, the cephalic end contains a few of them, while the middle of the ganglion is composed wholly of the very small ones. Some of the fibres of the radix longa seem to run through the ganglion without loss of their sheaths, while none of those of the radix brevis appear to do so. The radix brevis arises from the division of the oculomotorius for the internal and inferior recti muscles shortly after its separation from the trunk. The tiny twig from the first sympathetic ganglion joins the same division just before the radix brevis. The ciliary ganglion is drawn on Fig. 4 as if it lay on the dorsal side of the III nerve. It really lies on the lateral side. The ramus ciliaris brevis distally of the ciliary ganglion is drawn as if it contained only sympathetic elements. It may contain general cutaneous, though they could not be separately distinguished.

The mode of origin of the radix longa of the ciliary ganglion is somewhat different on the opposite side of this specimen. The first sympathetic ganglion is, as before, very intimately united to the Gasserian ganglion and the ramus ciliaris longus arises apparently from the sympathetic ganglion only. A small number of medullated fibres, which can be traced back through the Gasserian ganglion and are therefore probably trigeminal root fibres, can be traced from the Gasserian ganglion through the sympathetic ganglion and into the radix longa of the ciliary ganglion. These are accompanied in their course by a small number of large ganglion cells and after the separation of the radix longa from the sympathetic ganglion the number of these large cells is greatly increased.

There is thus formed a long, narrow ganglion made up wholly or nearly so of large cells like those of the Gasserian ganglion, which runs out along the radix longa and reaches from the Gasserian ganglion nearly to the ciliary ganglion. On this side the radix breva is much longer than on the left side and it arises from the undivided trunk of the oculomotor nerve.

Contrary to the statement of Stannius, the fibres of the radix brevis are exclusively of fine calibre, arising from a fascicle of fine fibres on the lateral side of the III nerve.

Distally of the ciliary ganglion the ramus ciliaris brevis lies laterally of all of the branches of the oculomotorius, ventrally of the m. rectus superior and dorsally of the m. rectus inferior and rectus internus. Opposite the optic chiasm it begins to turn ventrad and maintains this course, running under the optic nerve and above the m. rectus inferior until under the insertion of the latter muscle it penetrates the sclerotic and finally enters the iris at its extreme ventral point, almost diametrically opposite the entrance of the ramus ciliaris longus.

The ramus ciliaris longus (*cil. l.*) contains some fine fibres with almost as many very coarse and heavily myelinated ones. It follows the course of a small blood vessel ventrally of and parallel with the supra-orbital trunk and dorsally and somewhat laterally of the m. rectus superior. Dorsally of the eye it receives the anastomosing nerve from the second branch of the supra-orbital trunk. Then, without giving off any obvious branches, it penetrates the sclerotic and enters the iris somewhat caudad and laterad of the insertion of the m. rectus superior.

The arrangements of the ciliary nerves vary exceedingly in different fishes. For the details see Stannius ('49, p. 38) and Baudedot ('83, p. 123). The ramus ciliaris

brevis in most cases enters the eye-ball with or near the optic nerve. To this Menidia is a conspicuous exception.

The fact that fibres of the radix longa can be followed through the ciliary ganglion, while those of the radix brevis cannot, accords with the physiological results of Langley and Anderson ('92) and with the degeneration experiments of Apolant ('96; cf. also Huber, '97, pp. 124, 125).

Summary of Section 8.

The sympathetic nervous system so far as studied conforms in the main to previous descriptions, and most closely to Stannius' account of it in Belone. The "head part" contains six important ganglia, aside from the ciliary. These are related to the roots of the X, IX, VII, V and III nerves. The radix longa of the ciliary ganglion is accompanied by general cutaneous fibres which arise from a special projection of the Gasserian ganglion and were traced into the ciliary ganglion. These fibres I have homologized with the r. ophthalmicus profundus trigemini.

Section 9.—The Eye-Muscle Nerves.

I.—The Eye-Muscles.

The eye-muscles themselves merit a brief preliminary description. The sub-cranial canal is greatly developed and within it are the origins of the mm. rectus externus, rectus internus and rectus superior. It originates at the extreme caudal end of the basioccipital (Fig. 1, *s. c*), runs forward as a round canal at first within that bone, progressively expanding until farther cephalad it becomes broadly triangular in cross-section, the base of the triangle being dorsal. (Fig. 2). The floor of the canal is here

formed medianly by the parasphenoid, laterally mainly by the pro-otics, while the dorsal wall is for the most part formed by the pro-otics and by a projection from the basi-occipital bone. In the region of the figure just referred to the dorsal wall is membranous, a condition maintained from this point cephalad except at the extreme cephalic end, where a basisphenoid appears, just behind the optic chiasm. This bone is Y-shaped in cross-section, the lateral wings forming the floor of the brain between the hypophysis and the optic chiasm, and the vertical limb articulating below with the parasphenoid in the median line, thus forming the cephalic boundary of the sub-cranial canal.

Except at its most caudal end the canal is not filled by the eye-muscles, but these are packed densely with fat and connective tissue.

The m. rectus externus originates in the extreme caudal end of the sub-cranial canal, which at first it fills completely (Fig. 1, *r. e*). As the canal enlarges, this muscle rises to its dorsal side, while the m. rectus internus appears below it. With the lateral expansion of the canal still farther forward the externus occupies its dorso-lateral angle and here under the lobi inferiores a separate slip of the muscle takes origin from the fascia on the dorsal surface of the m. rectus internus in the median line, the fibres interdigitating with a similar slip from those of the opposite side (Fig. 22, *r. é*). There is thus formed a broad, thin sheet of muscle which runs dorso-laterally under a mass of fat to join the ventro-lateral side of the main muscular belly near the point where it leaves the sub-cranial canal (Fig. 2), turning abruptly laterad toward its insertion on the eye-ball. The fibres of this separate slip are very much smaller than the other

muscular fibres, a peculiarity which they exhibit from their origin to their insertion. They have, as we shall see, a slightly different innervation.

The m. rectus internus originates, as before indicated, near the caudal end of the sub-cranial canal, in a groove in the dorsal side of the parasphenoid bone. The muscles of the two sides occupy the ventro-median angle of the canal for its entire length (Fig. 2, *r. i. t.*) and still farther cephalad in the same relative position on either side of the parasphenoid along the inner sides of the orbits to their insertions on the cephalic borders of the eye-balls. The fibres which compose the dorsal edge of this muscle are of much smaller diameter than the other fibres.

The m. rectus superior originates in the cephalic end of the sub-cranial canal from the parasphenoid under the m. rectus internus and from the membranous roof of the canal over the same muscle. These two muscles run in close contact (Fig. 2) to the end of the canal, when the m. rectus superior turns dorsad and laterad running over the m. rectus inferior and the optic nerve to its insertion on the eye-ball. The fibres of the dorsal part of the muscle are smaller than the others.

The m. rectus inferior arises from the basisphenoid and runs over the m. rectus internus, under the m. rectus superior and the optic nerve to its insertion on the eye-ball. The ventral edge of this muscle is composed of smaller fibres than the others.

The mm. obliquus superior and obliquus inferior arise together far cephalad from the internasal cartilage running caudad to their insertions. In the centre of this massive cartilage a median unpaired horizontal canal (the anterior eye-muscle canal) is excavated, which runs from it caudal face cephalad for a considerable distance into its

substance and in which are the origins of these four muscles, two on each side, the canal being filled with the muscular fibres. The two obliqui superiores arise farther cephalad, the obliqui inferiores a little farther back from the dorsal wall of the canal. The latter lie nearer the median line and pass down between the superiores directly to their insertions on the ventral sides of the eye balls, each receiving a second slip of fibres *in transitu* from the ventral lip of the canal. The latter fibres are smaller than the others and run down the ventral edge of the muscle.

The obliqui superiores at the lips of the canal diverge and each receives a slip of fibres from the ventral lip which are, as in the former case, smaller, and which run along the dorsal edge of the muscle.

II.—The N. Abducens.

The sixth nerve arises by two fine roots, one .3 mm. caudad of the other. They arise mainly from a common nucleus of large cells which lies between them some distance from the median line and about two-thirds of the distance between the floor of the fourth ventricle and the ventral surface of the brain. There is a strong tract running transversely between the sixth nuclei of the two sides, but whether any of the root fibres have a crossed origin was not definitely determined. Another strong tract runs from each sixth nucleus dorsally through the overlying motor nucleus of the facialis and into the fasciculus longitudinalis dorsalis. Here again it is impossible to tell whether all or any of these fibres are root fibres, or whether this is a secondary tract. There are, however, two bundles of fibres, one on each side, which run up, one from each of the VI roots, laterally of the VI nucleus

and through the motor VII nucleus into the fasciculus longitudinalis dorsalis. These appear to be root fibres, thus putting the VI nerve directly into relation with the fasciculus.

Johnston ('98, p. 581) describes all of the VI fibres as arising from the fasciculus in Acipenser.

The sixth nerve at its exit from the brain contains some fine fibres mingled with the coarse ones. It has a short intra-cranial course mesially of the VIII nerve and dorsally of the lateral edge of the sub-cranial canal, which is here very wide. Under the lobi inferiores it passes ventrally through a foramen in the cranial floor into the dorso-lateral angle of the sub-cranial canal. Here it is crowded close against the lateral face of the m. rectus externus, which muscle a part of the fibres soon enter. Other fibres run to the ventral side of the muscle to enter the fine-fibred slip of the muscle which arises in the median line (Fig. 2). Of the latter nerve fibres, some of finer calibre run farther cephalad than any of the coarse fibres to supply these fine muscle fibres near their insertion upon the eye.

III.—The N. Trochlearis.

The nucleus of the fourth nerve is merely a caudal extension of that portion of the third nucleus (*q. v.*) which lies dorsally of the fasciculus longitudinalis dorsalis. The root fibres (mostly very large with a few smaller ones) pass up at once and cross over the mesocœle to emerge in the usual manner behind the optic lobes, after which they form a close round bundle closely applied to the inner face of the sensory V root (this part of their course not being shown on the plots).

Here the trochlearis root receives and carries for a

short distance a portion of the first root of the r. lateralis accessorius, as already described. After the separation of those communis fibres, it still contains about as many fine as coarse fibres (about 30 of each). The meningeal branches described by several authors as arising from the IV nerve in several types of animals probably are ultimately derived from similar anastomoses with communis roots. After the emergence from the cranium of the V+VII complex the trochlearis continues intra-cranially in the same relation to the brain as before.

At the level of the optic chiasm it pierces the cranial wall, which is here membranous, then, turning dorsad, it follows the outer wall of the cranium under the supra-orbital trunk, the finer fibres gathering on the dorsal side of the nerve. These relations maintain along the outer side of the internasal cartilage until the m. obliquus superior is reached, to the dorsal surface of which the nerve passes and into which it begins to send nerve fibres. The finer fibres, however, separate and run along the dorsal surface of the muscle to supply the smaller muscle fibres which occupy the dorsal edge of the muscle.

IV.—The N. Oculomotorius.

In conformity with the enormous size of the eye, the third nerve is also large. Its nucleus lies far dorsad near the median line, in part mesially of the large fasciculus longitudinalis dorsalis, but also, as it were, squeezed out so that a large part of it lies under the endyma of the floor of the mesocœle dorsally of the fasciculus and some also ventrally of it. Some fibres also plainly come from the fasciculus itself. Its origin being concealed in projection by the V + VII ganglionic complex, is not shown upon the plots.

Leaving the brain along the lateral face of the lobi inferiores, it passes at once through the cranial wall, which is here membranous, under the ventral edge of the sphenotic bone and mesially of the m. rectus externus. The m. rectus superior and the m. rectus internus lie farther ventrally. The III nerve contains at its exit from the brain chiefly the typical very large motor fibres; nevertheless there are mingled with these many of medium or small size, though all are heavily myelinated. The finer fibres tend to gather on the lateral aspect of the trunk and some of them go out with each of the rami. The larger part, however, enters the radix breva of the ciliary ganglion.

Under the cephalic end of the Gasserian ganglion it gives off first the branch for the m. obliquus inferior, then divides into dorsal and ventral portions, the former for the m. rectus superior the latter for the mm. recti internus and inferior.

The branch for the m. obliquus inferior pursues a rather peculiar course. Turning sharply ventrad and a little mesad and caudad, it runs down the cephalic face of the m. rectus externus from its dorsal to its ventral side, then curving around the outer side of the m. rectus superior from the dorsal to the ventral side of this muscle, it turns inward across the caudal face of the m. rectus inferior and around the ventral side of the m. rectus internus, closely wedged in between the latter muscle and the origin of the m. adductor arcus palatini. In this narrow space it crosses dorsally the r. palatinus without anastomosis. Having reached the parasphenoid, it turns dorsad between this bone and the m. rectus internus and then cephalad dorsally of the parasphenoid, mesially of the m. rectus internus and close under the optic chiasm, the

nerves of the two sides lying close together, almost in contact with each other. At this level the rami palatini of the two sides pursue very similar courses, but on the ventral side of the parasphenoid. Cephalad of the chiasma it turns dorsad, still in the median line, and continues forward close under the brain and olfactory nerves. Upon the appearance of the internasal cartilage it begins to diverge from the median line and soon turns ventrad to enter the m. obliquus inferior. It applies itself to the dorsal surface of the muscle and the coarse fibres enter the belly of the muscle in many strands, while the finer fibres separate and run down among the finer fibres of the muscle nearer their insertion.

The nerve for the m. rectus superior immediately after its separation from the oculomotor trunk divides into a dorsal portion of fine and medium fibres and a ventral coarse-fibred portion, both of which turn dorsad and apply themselves to the ventro-lateral face of the muscle. The coarse-fibred portion at once enters the belly of the muscle; but the finer fibres follow along the smaller muscle fibres of its dorsal edge, occasionally sending twigs into its substance, to its insertion upon the eye-ball.

The remainder of the III nerve gives the radix breva to the ciliary ganglion and at once divides into rami for the mm. recti internus and inferior. The former runs directly cephalad under the m. rectus superior and over the mm. recti internus and inferior. Crowded ventrally by the emerging optic nerve, it crosses the cephalic face of the last mentioned muscle and enters at once the m. rectus internus. The coarse fibres bury themselves in its substance, but the finer ones run along the dorsal border or embedded in a mass of exceedingly small muscle fibres which occupy the dorsal part of the muscle, in which

relation they can be followed to the insertion of the muscle upon the eye-ball.

The nerve for the m. rectus inferior after separation from the nerve last described descends at once to the dorsal surface of its muscle and here breaks up into numerous branches. Of these those with coarse fibres enter the belly of the muscle, while the fine fibres run to the small fibred ventral edge of the muscle, which they follow to the insertion upon the eye-ball.

V.—Critique of the Eye-Muscle Nerves.

In the case of each of the six eye-muscles of which we have just been treating, the side along which the finer fibres of its nerve run contains much smaller muscle fibres than those which make up the body of the muscle, the diameter of these small muscle fibres often being no greater than that of a large nerve fibre. The smaller muscle fibres are not merely the ends of larger ones which have become attenuated near their insertion, but they run for nearly the whole length of the muscle, maintaining the same diameter and the same relation to the larger ones. They do not appear to differ from the ordinary fibres except in size, in their constant relation to the finer nerve fibres and particularly in the fact that they are in places more closely enveloped by a dense and very rich plexus of these finer nerve fibres and by a nucleated connective tissue interstitial substance. The investigation of the nerve endings here by proper methods might yield interesting results.

That the small muscle fibres and the small nerve fibres are related can scarcely be doubted. The source of the small nerve fibres could not be certainly determined. The most natural supposition is that they come from the

fasciculus longitudinalis dorsalis, though this conjecture could not be verified. We already know that the oculomotor nuclei are placed in relation with each other and several sensory centres through the mediation of this tract. Cf. the diagram of Bonnier, '95. And this would suggest that possibly we have here a sensory mechanism analogous with the muscle spindles. The structure, however, does not conform very well, and we are told, moreover, that spindles do not occur in the eye-muscles (Batten, '97, p. 176). The comparison is rendered more difficult by Sherrington's experimental proof (fide Huber and DeWitt, '98) that the spindle nerves are derived from the dorsal spinal roots, while the small oculomotor fibres quite certainly come out with the other and undoubted motor fibres of the eye-muscle nerves. The whole question demands further study.

In order to facilitate comparison of the eye-muscle nerves of Menidia with Allis' account and the elaborate phylogenetic scheme which he has elaborated ('97), I have prepared the diagram, Fig. 13, which should be compared with Allis' Fig. 12, Plate XXII.

The courses of the trochlearis and abducens are in Menidia essentially as in Amia; but the relations of the oculomotorius are in several respects different. This nerve emerges from the cranium some distance ventrally of the Gasserian ganglion and is crossed externally while still within the foramen by the truncus infra-orbitalis. Its foramen is far caudad of that of the optic nerve and at the level of the latter all of the branches of the third nerve except that for the m. rectus superior lie ventrally of the chiasma. In all of Allis' figures the ophthalmic nerves (superficial and deep) arise from a common stem which lies ventrally of the III nerve, and he states in the text that in Amia the latter nerve "pierces the lining membrane of the cranial cavity opposite and above the

optic chiasma." In Menidia, too, the branches do not leave the oculomotorius in the same order as in Amia, or any other form figured by Allis, the nerve for the m. obliquus inferior being the first instead of the last branch to be given off. This nerve passes under the inferior and internal recti, as in Allis' figures, but the main nerve, viz., the portion for the ciliary ganglion and for the inferior and internal recti, lies above these muscles. Allis' conjecture that the arrangement in Amia is typical for all ganoids and teleosts certainly will not hold, so far as the teleosts are concerned, at any rate.

Allis points out certain errors and ambiguities (due in part to misplaced reference letters) in my account of the eye-muscles in Amblystoma ('94). I have re-examined these nerves and the correct relations in Amblystoma are as follows (cf. Fig. 14):

The IV and VI nerve require no additional comment save that they conform to Allis' diagram of the Anura and not to his diagram of the Urodela.

The foramen of the III nerve is a little caudad and very slightly dorsad of that of the optic nerve. Immediately after its exit from the foramen the oculomotorius lies just laterally of the emerging optic nerve and mesally of the mm. recti superior, inferior and internus, near their tendinous origins. Just laterally of these muscles is the r. ophthalmicus V, all of these structures lying at very nearly the same dorso-ventral level. Here the III nerve divides into its dorsal and ventral rami, the former passing to the m. rectus superior only. This branch and its muscle lie dorsally of the ophthalmic nerve and the latter crosses the n. opticus dorsally.

The ventral branch of the III nerve turns down behind the optic nerve and crosses the latter on its ventral side, turning laterally and crossing over the m. rectus internus very near its tendinous origin. Here it lies crowded closely between the rectus internus and the dorsal edge of the rectus inferior, the former lying mesally, the latter laterally of it. In some cases a few fibres seem to enter the rectus internus at this point. It then enters the

dorsal edge of the rectus inferior and within that muscle divides, some fibres remaining within the muscle and others emerging on the cephalic and ventral side of it. The latter soon separate from the muscle and continue cephalad. A branch is given off which runs dorsad to supply the rectus internus and the remainder runs far forward to supply the obliquus inferior.

The oculomotorius, then, runs under the m. rectus superior, the r. ophthalmicus and the n. opticus. It crosses the m. rectus internus dorsally near its origin, but lies far ventrad of that muscle when the main nerve for it is given off. It pierces the dorsal portion of the m. rectus inferior and enters the obliquus inferior from its dorsal side.

Now, it is obvious that this arrangement in Amblystoma conforms to the scheme which Allis gives for the Anura, rather than the one for the Urodela. The lack of uniformity of my results with his scheme, both in the fishes and the Amphibia, together with the fact that his diagrams do not in all cases correspond with his authorities (*e. g.*, in the diagram of the Cyclostomata, based on Fürbringer, the nerve to the m. rectus superior is drawn over the ophthalmicus profundus, while Fürbringer says that it runs below that nerve) suggest that his entire phylogenetic scheme should be received with some reserve.

Again, to say nothing of the acknowledged incompleteness and possible inaccuracy of the data upon which the table is constructed, it seems rash to construct even a tentative phylogenetic tree upon a single character of this sort. But, aside from this, unless one were to apply the neuro-muscular theory rigidly in the ontogeny (which few morphologists now-a-days are willing to do) it is difficult to see why so exaggerated importance should be given to the relative positions of these nerves and muscles. There is no sufficient evidence that these nerves have been split off from the skin, as Allis assumes that most of the cranial nerves have been (a point to which we shall recur), but on the other hand, the best recent work on this subject (*e. g.* Dixon, '96 and Neal, '98) adds very emphatic testi-

mony to the doctrine that the eye-muscle nerves grow directly out from the brain. If this be true, I see no reason why a given motor nerve should not grow out either above or below some other structure, depending upon the peripheral relations of its end-organ with reference to that structure.

Finally, Allis in his account of the elasmobranchs (p. 522) says: "These different relations of the oculomotorius to the internal and superior recti, in elasmobranchs, are due to and are caused by the gradual shifting from before backward of the origins of all the recti muscles, and also of the place of exit of the oculomotorius from the cranium. As a result of this shifting the internal and superior recti, at their origins, either traverse, or are traversed by, the issuing nerve." That is, he invokes a principle to account for the diverse relations of nerve and muscle in elasmobranchs, which, if applied more broadly, might weaken the phylogenetic value of some of his other cases.

It seems to me, therefore, that Allis' phylogenetic table is based largely upon inconclusive data, and that the various arrangements which he diagrams are of cenogenetic origin rather than phylogenetic significance. In the case of Menidia the deviations from what Allis regards as the typical piscine arrangement can be easily explained mechanically by the great size of the eyes and the consequent crowding of the recti muscles far backward.

VI.—Summary of Section 9.

The sub-cranial, or eye-muscle canal is very highly developed, running back under the cranium for its entire length to the caudal end of the basioccipital bone. Correlated with the large eyes, the eye-muscles are highly developed. The recti, except the rectus inferior, arise in the sub-cranial canal. The obliqui arise in an anterior eye-muscle canal in the internasal cartilage. All of the eye-muscles have some smaller muscle fibres which usually

originate from a different position from the others and are innervated by smaller nerve fibres. Their significance is unknown. All of the eye-muscle nerves derive most of their fibres from their nucleus of the same side, but some from the nucleus of the opposite side and doubtless some from the fasciculus longitudinalis dorsalis. The eye-muscle nerves of Menidia do not conform to the scheme given by Allis for the ganoids and teleosts, and an examination of his whole system leads to the conviction that its phylogenetic value is greatly in need of confirmation.

Section 10.—The Optic Nerve.

This nerve is enormous and is composed of very small fibres. The chiasm is the simple crossing with the left nerve uppermost which is typical for teleostomes. The cranial wall at the point of emergence is membranous. The nerve is of the broad, plicated, ribbon-shape so common among the bony fishes, consisting of three laminæ. One septum enters from the dorsal side, the other from the ventral. The adult structure certainly favors the view of Studnicka ('96) that this form is derived by the folding of a ribbon-shaped nerve, rather than that of Deyl ('95) that it is derived from a cylindrical nerve by the intrusion of connective tissue septa. This form must be regarded as an adaptation to secure the proper nourishment of the nerve in these large-eyed forms, as Studnicka points out, and Deyl's attempt to deduce phylogenetic conclusions from the forms of the optic nerve can hardly have much value, as the character is too variable and too liable to convergence. For further morphological considerations see the papers cited above.

Section 11.—The Olfactory Nerve and Nasal Organ.

The olfactory bulbs are in part overshadowed by the cerebrum. The bulb gradually tapers into the cylindrical olfactory nerve, which penetrates the membranous wall of the cranium and then continues cephalad along the lateral face of the internasal cartilage. As the latter begins to expand around the nasal sac, the nerve is crowded under the m. obliquus superior near its origin and then penetrates the internasal cartilage through a foramen which is in part lined by a V-shaped projection from the parethmoid bone. Having reached the olfactory fossa, it runs along the inner side of the non-sensory part of the olfactory sac, being separated from it, however, by a great lymph sinus. Here it breaks up into numerous branches which enter the lamellæ of the sensory portion of the sac.

The nasal organs of Menidia are large and well developed, like the other organs of special sense. The anterior (cephalic) opening is a very small pore (*n. a. a.*), lying laterally of and close to the cephalic tip of the supraorbital canal. This pore opens directly into the cephalic end of a wide sac containing three large lamellæ, which are attached to the ventro-mesal wall of the sac and which extend lengthwise for almost the entire length of the latter, and two smaller lamellæ at the cephalic end of the sac. These lamellæ and the walls of the sac adjacent (but not the dorso-lateral wall of the sac) are thickly covered with the bud-like groups of sensory cells so characteristic of the teleosts (Blaue, '84).

These "olfactory buds" are closely packed in the mucous membrane along the whole surfaces of the lamellæ except at their tips, very much like Blaue's figure of Trigla, though not with so great regularity as his drawings would indicate. The "olfactory buds" vary in size,

the largest being half the size of the largest naked cutaneous sense organs found on the outer surface of the head.

Passing caudad, shortly before reaching the posterior nasal opening the lamellæ disappear and with them the sensory epithelium, the entire sac from this point caudad being lined by ordinary thin pavement epithelium. The sac narrows in its transverse diameter and at the same time becomes much deeper. It extends as a rather narrow cleft so far ventrally that its deepest point lies in the same horizontal plane as the dorsal ends of the dorsal diverticula from the lateral edge of the pharyngeal roof, these diverticula lying somewhat laterally of the olfactory sac. The outline of the nasal sac is drawn on Fig. 5, the sensory portion of the wall being shaded. The posterior nasal aperture is a long narrow slit four or five times the length of the anterior. The nasal sac extends only a very short distance caudad of it. For further notes on the conformation of the olfactory fossa see the account of the terminal portion of the supra-orbital trunk, Section 7, IX.

The development of the olfactory organ has been worked out in a series of post-embryonic stages and I fully confirm Madrid-Moreno ('86) that the ontogeny disproves the elaborate assumptions of Blaue ('84) that the olfactory epithelium is derived from a bud of the lateral line system which has wandered into the olfactory fossa, there multiplied to form the system of "olfactory buds" of the teleosts and then, in most higher forms, formed secondarily a continuous sensory surface by the fusion of the buds.

From the standpoint of theoretical morphology alone Blaue's results could not stand; but, as it is a question of no small importance to the fundamental head problems, and as Blaue's errors reappear in recent editions of several

of our standard text-books (*e. g.*, Minot's and Hertwig-Mark's Embryologies), it seems desirable to emphasize the matter again.

Section 12.—General Considerations.

I.—The Ramus Lateralis Accessorius.

This nerve is the r. recurrens facialis, the r. lateralis trigemini and the superficial lateral line nerve of the literature. It and related nerves have also received many other names. By previous writers it has very generally been regarded as morphologically analogous with the r. lateralis vagi. It is, however, now known that this is not the case and the term lateralis accessorius has hitherto been avoided by me as suggesting bad morphology. The reasons, however, given by Cole ('98a) for the retention of this good old term of Weber's seem to me to be sufficient; it is merely necessary to keep constantly in mind that this nerve has no morphological relationship whatever with the r. lateralis vagi.

This nerve arises by two root complexes, one from the vagus system, the other from the facial. These anastomose above and behind the cranium and the combined nerve runs back into the trunk near the dorso-median line. The most interesting morphological question in this connection is the problem of the relation of this nerve and the organs of the accessory lateral lines supplied by it to the r. lateralis vagi and the main lateral line. As I have before remarked, all of the cutaneous sense organs are somewhat reduced in Menidia and especially the terminal buds, so that this is not a favorable type for the solution of this problem. In effecting the analysis of these nerves we must rely mainly upon the calibre of the nerve fibres and the problem is complicated greatly in this species by

the fact that the reduction in size of some of the organs of the lateral line has involved the reduction also of the calibre of the nerve fibres which supply these organs, so that the normal distinction between lateralis and communis fibres is in a measure obscured. Nevertheless, I think that the conclusions as expressed on the plots are correct.

I am convinced that in Menidia the r. lateralis vagi does not innervate any organs other than those of the lateral line. The participation in the general innervation of the skin or muscles can be excluded. The fibres from this nerve which anastomose with the r. lateralis accessorius are much finer than any lateralis fibres. In a few cases fibres from these anastomosing nerves supply naked cutaneous sense organs. These fibres, too, are finer than the lateralis fibres and I regard them and the anastomosing fibres as both belonging to the communis system. They may be derived from the lobus vagi by either one or both of two paths: (1) The fine fibres already mentioned as emerging from the brain in the lateralis root enter that root on its caudal side and may be derived from the lobus vagi, though they could not be traced back into it. (2) The anastomosing fibres from the root of the glossopharyngeus to the lateralis root are almost certainly communis fibres and I look upon this tract as the probable source of all of the fibres under consideration. These fibres, as they separate from the IX root, are rather larger than the other fibres of that root but not nearly so large as the proper lateralis fibres, *i. e.*, they are about the same size as the fibres of the branches of the r. lateralis which I have designated as belonging to the communis system on the plots.

The dorsal cutaneous rami of the vagus have been

already partially treated (under Section 5, VIII). These may comprise the general cutaneous rami for the skin, as described by the older writers, and communis fibres for terminal buds, as described by Allis ('97), besides the ramus supra-temporalis proper for the lateral line organs (see Section 5, X, 1). The three classes of fibres may be variously fused. In Menidia we have seen that the general cutaneous fibres arise separately from the jugular ganglion and pursue their devious ways to the skin without at any point coming into relation with the lateral line nerve. It is improbable that any fibres, except lateralis fibres, enter the r. supra-temporalis vagi, save the communis component from the anastomosing ramus from the IX root.

From the rather inharmonious accounts of Stannius and Baudelot it is clear that fusions of other sorts may occur in the bony fishes. In Fierasfer, Emery ('80) describes the r. supra-temporalis vagi (lateralis) and the r. opercularis vagi (general cutaneous) as fused. In Amia (Allis, '89, p. 518), the r. supra-temporalis vagi (lateralis system) and the ramus cutaneous dorsalis are distinct, though closely associated. The latter probably contains both general cutaneous and communis fibres.

The r. supra-temporalis X and the r. supra-temporalis IX, both lateralis nerves, may be present at the same time (Amia, Allis '89 and '97) so that these cannot be regarded as the same nerve, sometimes going out with the X nerve, sometimes with the IX. Both are present also in Læmargus, as we learn from Ewart and Cole ('95, p. 475). It is a most remarkable fact that according to their description the r. supra-temporalis vagi supplies the organs of the occipital commissure and several organs of the main line cephalad of it, while the supra-temporalis IX

supplies the three organs of the main line immediately caudad of the commissure. This is a condition not known for any other vertebrate and indeed Ewart himself had predicted ('93, p. 72) that if the IX nerve should prove to supply any portion of the lateral line of the head it would be the pre-commissural, not the post-commissural portion.

The fibres which I have described running from the IX root to the lateralis root have an exact counterpart on a larger scale in Amia (Allis, '97, p. 625). They are described as coming from the ventral, *i. e.*, communis, portion of the IX root, and not from the dorsal, or lateralis portion. What the fate of these fibres is peripherally in Amia, has not been determined. It is natural to assume that they supply either terminal buds of the trunk or accessory lateral line structures.

Allis ('97) assumes, I think correctly, that the terminal buds scattered over the bodies of many fishes, (*e. g.* gadoids) are innervated by the r. lateralis accessorius, which is distributed to those regions. There are, however, in some fishes extensive regions of the body not reached by this nerve which are known from the researches of Leydig and others to be abundantly supplied with terminal buds, which are said to be innervated by the r. lateralis vagi. Now, if this passage of communis fibres into the r. lateralis, as we find it in Menidia, is of general occurrence, it clears up a serious difficulty in the interpretation of these terminal buds. We have seen that such is the case in Amia and from Allis' account ('89) it would appear that the anastomosis is there much larger than in Menidia.

I predict that in cyprinoids, where the lobus vagi is so large, and the body is known to be covered with terminal buds (Leydig, '94) that these will be found to be supplied

from it via the r. lateralis vagi and other nerves and that in siluroids and gadoids terminal buds of the same regions of the trunk will be supplied from the fasciculus communis via the r. recurrens VII, or r. lateralis accessorius.

The different accounts of the anastomosis between the VII and the IX and X nerves present a very interesting series.

In the cyprinoids and in Gadus merlangus, Baudelot ('83, p. 129) describes an intra-cranial communication from the V (VII?) nerve to the IX. In the former case it is large, runs internal to the ear and VIII nerve and anastomoses with the IX and X nerves and then forms a recurrent branch for the trunk which anastomoses with the first spinal. In Gadus merlangus it is very small and runs internal to the ear, but external to the VIII nerve, with which it anastomoses. He regards the cases as homologous and from a comparative study of a number of cyprinoids concludes "that even in the cyprinoids, the recurrent bundle exhibits a tendency to rise up on the side of the medulla in such a way as to stride over successively, so to speak, each of the nerves which springs from this part of the medullary axis." In Gadus merlangus this process is carried a step farther and we may carry this series even farther than Baudelot has done, to include the extra-cranial anastomoses such as I have described in Menidia.

The intra-cranial communicating branch between the vagus and the "r. lateralis V," which Stannius, Baudelot and others describe in many fishes, is totally wanting in Menidia, as in Silurus, ('49, p. 50), as would be expected if these recurrent nerves all belong to a single system whose position may vary from an intra-cranial anastomosis with the IX and X nerves to a sub-cutaneous anastomosis

with the same nerves. The cases all have this in common, that they are related centrally to the communis system of nerves and peripherally supply the meninges and skin of the top of the head and of the trunk. It appears from the descriptions of Merkel ('80), Leydig ('94), Harrison ('95), Allis ('97) and others that the cutaneous distribution is mainly, if not wholly, to terminal buds.

The relations to the vagus system are most various. In most, if not all, of these cases it is clear that communis fibres with essentially the same distribution go out with the IX or X nerves or both and the anastomosis of these two sets of fibres is easily explicable. Since the branches which go into the body supply in some of the cases terminal buds in the same regions as the lateral line organs supplied by the r. lateralis vagi, the more or less intimate anastomosis with the latter nerve is also easy of comprehension.

Phylogenetic speculations are, perhaps, premature, yet from the evidence now in hand I incline to the belief that the peripheral anastomosis is the more primitive. As terminal buds migrated into the trunk from the head, communis fibres seem to have accompanied them from both the VII and IX roots and probably also the vagus. These nerves effected sub-cutaneous anastomoses with each other and probably with similar fibres accompanying the r. lateralis vagi. Menidia, then, is probably very near the primitive type. As the recurrent systems increased in importance, two lines of differentiation were followed. On the one hand, the facialis portion was exaggerated at the expense of the post-auditory portion and we have forms like the siluroids with enormous r. recurrens VII with no considerable vagal participation. In

Amia the facialis portion has been reduced almost beyond recognition, being represented only by a delicate anastomosis between the terminal filaments of the first branch of the r. ophthalmicus superficialis V and dorsal cutaneous twigs of the vagus (Allis, '97, p. 600). The post-auditory portion, however, is represented by a large communis element which passes from the IX root to the lateralis root of the vagus and whose distribution is evidently very much like that of the corresponding, but smaller, nerve in Menidia. In forms like the gadoids the superficial recurrent nerve from the VII nerve is of considerable size and it is joined intra-cranially by a small post-auditory portion from the vagus. And finally in some of the cyprinoids the external (superficial) element from the facialis has been altogether lost and the anastomosis from the VII to the IX + X is altogether intra-cranial.

Since the preceding pages were written I have received Cole's paper ('98a) to which reference has already been made, and I am pleased to find that my conclusions confirm in most important respects those of this eminently careful student. In some points regarding the morphology of the r. lateralis accessorius and related structures, however, I cannot follow him. All that could be gained by dissection has been done and well done. The microscopical anatomy, however, was done on sections of very young codfish, which must have been poorly adapted to the purpose, for he did not succeed in his analysis of the trigemino-facial roots.

The conclusions to which he was led, both in his examination of the literature and in his study of the sections, are in some important respects so different from my own that I have been led to examine the condition in Gadus. As this investigation is still unfinished at the time when

these sheets pass through the press (July, 1899), I shall here omit the somewhat extended critique of Cole's work which I had originally prepared, and content myself with the statement that I do not confirm his findings with reference to the geniculate ganglion and the facial root of the r. lateralis accessorius of the cod.

In brief, the relations of the geniculate and sympathetic ganglia are almost exactly as in Menidia, though the whole trigemino-facial complex is much more compact in Gadus. The geniculate ganglion is wholly intra-cranial and so closely joined to the Gasserian ganglion that Cole failed to differentiate them and mistook the extra-cranial sympathetic ganglion for the geniculate. The root of the r. lateralis accessorius arises wholly from the geniculate and not at all from the Gasserian, just as in Menidia. The details of these connections in Gadus with full illustration will be published shortly.

A word further upon the question of "collector" nerves. Of the longitudinal nerve trunks running through the body the sympathetic chain, with its anastomosis with every spinal nerve, is the best illustration of a true collector nerve. The older writers have frequently described the r. lateralis vagi as a similar collector, supposing that it receives accessions from each spinal nerve through the r. medius. This, we now know, is not the case, as there is no anastomosis here such as would justify us in regarding the r. lateralis as a collector. There remains to be considered the r. lateralis accessorius. Stannius lays great stress (p. 151) upon this nerve as a collector of all spinal and spinal-like nerves. In view of its function as the nerve supply for the dorsal row of terminal buds, it is not probable that its primary form was that of a collector; nevertheless its uniform anastomosis with the spinals is to be explained.

It is possible that in the case of both of the lateral line nerves the relation to the spinals is purely accidental and due to the fact that the paths of the nerves in question cross in the inter-muscular septa. This seems especially probable in the case of the n. lateralis vagi from its course in the septum between the dorsal and ventral musculature and from the loose and variable nature of the anastomosis. The relation of the r. lateralis accessorius in the septum between the dorsal musculature and the interspinal muscles is similar. Nevertheless the remarkable constancy of these anastomoses, especially in the latter case, lends credence to the belief that they have a morphological and a physiological basis. Stannius was certainly correct (p. 151) in rejecting the morphological equivalency of the dorsal series of anastomoses related with the r. lateralis accessorius and those of the ventral rami with the sympathetic chain. Nevertheless they may have certain physiological features in common which will justify a comparison.

All of the viscera of the trunk and head seem to be intimately related to each other and to the central nervous system through the mediation of the sympathetic nervous system. Now the communis system, as we have used that term, contains in addition to fibres for terminal buds other sensory fibres which we have termed *visceral*. What may be the relation (if any) between these visceral fibres and those of the sympathetic cannot perhaps be determined at present; but, like the sympathetic, they are known to have a wide distribution to exposed visceral surfaces, and I think also to the internal parts as well. In short, they participate in the general sub-cutaneous and internal plexus which enables the body to react as a physiological unit. The enormous physiological significance of this universal plexus is coming to be better appreciated than

formerly; and it has a morphological value which is not as yet fully recognized. The fibres of this plexus will tend to gather about and distribute with the larger nerve trunks of whatever type simply because these afford the paths of least resistance; and if two nerves cross, even though they be of unlike composition and do not exchange fibres with each other, yet this will form a nodal point in this plexus of visceral fibres. This factor will operate to draw contiguous nerves together, even though they may be of totally dissimilar composition, and may account for the tendency, everywhere noticeable, for nerves of the several components to accompany each other, often coming from widely separated origins and deviating from their natural courses to do so.

The ramus lateralis accessorius originates in Menidia in an elaborate plexus of communis fibres, arising partly from the facial nerve and partly from the vagus complex. This plexus is exceedingly rich and intricate in the meninges and under the skin of the dorsal part of the head and trunk, only the main nerves being indicated on the plots. This nerve was, doubtless, primarily visceral in its proximal portions at least, while in that portion which leaves the head to enter the trunk I incline to the belief that the terminal bud fibres appeared first. But be that as it may, the visceral fibres followed and when the r. lateralis accessorius crossed the dorsal rami of the spinal nerves, connections of their visceral fibres were secondarily effected. In cases where the r. lateralis accessorius sends a ventral branch to the anal fin, as in Gadus, that branch effects similar connections with the ventral rami of the spinal nerves.

The r. lateralis vagi may effect similar quite secondary connections with the r. medius of the spinal nerves; but,

as the latter rami do not bear so constant a relation to the inter-muscular space laterally of the dorsal musculature as the dorsal rami to the space mesially of it, their anastomoses with the r. lateralis are neither so extensive nor so constant as the others.

II.—Metamerism.

Certain questions of metamerism and the relations of the components to each other remain to be considered.

Our conception of the composition of the primitive segmental nerve will depend somewhat upon whether we regard the cranial (branchiomeric) or the spinal type as the more primitive. But in either case we may assume with great probability that the typical segmental nerve in the earliest vertebrate contained somatic sensory, or general cutaneous fibres, also somatic motor for the voluntary musculature and viscero-motor and viscero-sensory components. In the head the form which the segmental nerve takes is dominated by the branchiomerism, and we are justified now in concluding that the original branchiomerism coincided with the metamerism (see especially Neal, '98).

We have suggested in the preceding sections the following comparisons between the components of the head and those of the trunk. The general cutaneous and somatic motor of the cranial nerves are strictly homodynamous with the corresponding components of the spinals. The viscero-motor of the trunk is present in the same form in the head, but has also suffered an extreme differentiation and very profound modification in connection with the branchio-motor apparatus. The rudimentary visceral sensory system of the trunk is represented by the communis system of the head and has also suffered great

specialization and modification, in this case in connection with the sense organs developed in the mouth and secondarily in the outer skin. The acustico-lateral system is evidently a later acquisition developed perhaps from the general cutaneous system, perhaps from the terminal buds.

All of the components which I have enumerated were therefore probably present in the typical primary branchiomeric nerve, except those for the specialized sense organs, lateral line organs, taste buds, terminal buds, and, of course, the organs of higher sense, eye, ear and nose. The fibres for these specialized organs appeared sporadically as evoked by the physiological requirements of each case, and each, for obvious physiological reasons, is related centrally to a single centre. In the case of the cutaneous sense organs especially, these requirements have been exceedingly variable. Thus the lateralis fibres converge toward the tuberculum acusticum from either side of the auditory capsule. The relations of the ganglia and roots of these fibres are quite constant throughout the fishes, as also are the principal nerve trunks, such as the r. lateralis vagi, the r. ophthalmicus superficialis VII, the r. buccalis and the r. mandibularis externus VII. But it is quite different with the smaller lateralis branches, such as the r. oticus, the r. supra-temporalis, etc. The courses which these may take will depend partly upon the arrangements of the corresponding organs and partly upon mechanical convenience growing out of the disposition of adjacent organs, particularly other nerves, for it is a general rule that two nerves which pass near each other, whatever may be their composition, tend to fuse into a common trunk. In the same way communis fibres from terminal buds may accompany any of the cutaneous

nerves, depending upon the arrangement of these organs.

Hence, the arrangement of the components in its main outlines, including the terminal centres of the special sensory systems, is a palingenetic character of great permanence throughout the vertebrata, which is not due to the direct influence of the now present environment. On the other hand, the peripheral courses of these nerves, the number of rami, their individual composition, anastomoses and fusions are to some extent cenogenetic characters to be explained by the pressure of the environment, mechanics of growth, etc.

If, now, we frame a conception of the typical spinal nerve of the existing Ichthyopsida, as illustrated by the bony fishes, we find that it receives all of the four primary components and that each of its rami also receives some fibres of each of these four categories. Thus the ventral ramus of the spinal nerve contains considerable numbers of somatic motor, somatic sensory, viscero-motor and viscero-sensory fibres. The two last components reach their peripheral distribution partially and perhaps wholly through the mediation of the sympathetic nervous system. The medial and dorsal rami contain the two somatic components in large numbers and the two visceral components in very much smaller numbers, chiefly in the form, no doubt, of vaso-motor and excito-glandular fibres with the corresponding return reflex paths.

The central connections of the visceral fibres, and especially of the viscero-sensory fibres in the spinal cord are very obscure. Johnston ('98, p. 597) denies the presence of any viscero-sensory fibres in the spinal nerves. "No sensory fibres of the spinal nerves supply visceral structures. We know of no sensory fibres entering the spinal cord from the sympathetic system." Kölliker ('96, p. 860) makes a similar statement,—"The sensory fibres of the sympathicus all arise from cerebro-spinal fibres and end, like cerebro-spinal sensory elements, in the periphery. The sympathicus possesses no sensory

fibres properly its own." This view is not without opponents. Onuf and Collins ('98) find degenerations after extirpation of sympathetic ganglia which they can explain on no other assumption than that of sensory fibres running from cells of the sympathetic ganglia into the dorsal roots.

But leaving the question of the sympathetic connections to one side, we have the most abundant evidence (see *e. g.*, Thane, '95, p. 350, Kölliker, '96, p. 858, and Huber, '97, p. 131) that sensory cerebro-spinal fibres distribute freely among all of the viscera through the mediation of the sympathetic nerves. The findings of experimental physiology and pathology also necessitate the assumption of such fibres in order to explain the phenomena of the reflexes, etc. Where the spinal centres for these visceral fibres may be has not been accurately determined, but the presumption, it seems to me, is rather in favor of than against the idea that they are distinct from the general cutaneous centres. It is, undoubtedly, true that this spinal viscero-sensory system is very small and that it is not present in all of the spinal segments is very probable; for, as we have seen, the functions of this system have been very largely usurped by the cranial visceral system of the vagus.

Passing now to the head, the presence of the branchial apparatus and the reduction of the somatic musculature have so modified the conditions as to render comparison with the trunk almost impossible. If we exclude the twelfth cranial, which in the fishes is but little modified from the typical spinals, there is no cranial nerve which has a ventral ramus of the typical form. The reason is clear. There is no ventral somatic musculature in the head aside from that which has grown forward from the region of the first spinal. And in the higher fishes the overgrowth of the operculum has eliminated in the branchial region the cutaneous areas which would be innervated from ventral spinal rami. It would be interesting to learn in the sharks the exact details of the innervation of the skin of the venter in the gill region. The branchial trunk is commonly supposed to represent a ventral ramus. Whether this is so can only be determined positively after a more careful examination

of its composition in the lower fishes. If this proves to be the case, it is clear that in the higher fishes it contains, for the reasons already mentioned, only the two visceral components.

The lateral line branches of the cranial nerves have usually been considered equivalent to the lateral or medial rami of the spinal nerves. Thus, the r. lateralis vagi has often been described as a "collector" of these medial rami, and very recently both Fürbringer ('97) and Neal ('98, p. 271 and p. 211) consider that these medial rami have been "supplanted" by the r. lateralis vagi. To this there are at least two very grave objections. In the first place, these medial rami have not been supplanted at all in the bony fishes but they, and they alone, innervate all of the skin and all of the muscles of the dorso-lateral regions of the body. Neither the r. lateralis X nor the r. lateralis accessorius participate in the innervation of these cutaneous areas, but supply only special sense organs which have migrated in the ontogeny back from the head. And from this it follows, in the second place, that these recurrent nerves would be incapable of supplanting the dorsal or medial spinal branches, for they are not of equivalent structure or function. This case is totally different from that of the r. intestinalis, where there has been a supplanting of visceral spinal fibres by visceral cranial fibres.

In the same way we must avoid homologizing such dorsal cranial nerves as the r. supra-temporalis, composed of lateralis fibres, with the general cutaneous fibres of the dorsal rami of the spinal nerves. The rami cutanei dorsales of the vagus and the r. ophthalmicus superficialis V (and possibly the general cutaneous fibres with the r. oticus) are the only nerves in the head of Menidia which can be homologized with dorsal spinal rami, though in

other forms such general cutaneous fibres may be present in other cranial nerves, as in the ninth.

We may now, perhaps, attempt to formulate a scheme to express the typical branchiomeric nerve of higher fishes. This nerve has a dorsal ramus to the skin, which contains general cutaneous fibres and probably a few visceral fibres. Motor somatic fibres are absent on account of the loss of the dorsal musculature. The remainder of the typical nerve is contained in the branchial trunk, which is composed of visceral sensory and visceral motor fibres. From this trunk is given off a palatine branch, which is all sensory, and a pre-trematic branch, which is also sensory, and a post-trematic branch which is mixed.

Now, as the special sense organs are differentiated, fibres from taste buds will come in by way of the palatine and the pre- and post-trematic branches, and other communis fibres from terminal buds on the outer surface of the body may come in with the general cutaneous fibres of the dorsal ramus. With the appearance of the lateral line organs, their fibres may also enter with the dorsal rami, though most of them appear to come in as independent trunks. The latter may represent medial rami in which the general cutaneous fibres have disappeared, or, more likely, they have no representative in the spinal nerves.

This scheme applies in the post-otic branchiomeres, where the growth of the operculum has involved the reduction of the ventral cutaneous areas. In the pre-otic branchiomeres these relations are disturbed by the differentiation of the jaws and of the facial skeleton in general, and also by the exaggeration of the dorsal cutaneous areas, since the whole operculum is morphologically a dorsal or dorso-lateral structure belonging to the facial segment.

In the facial segment the post-trematic ramus has the typical communis and viscero-motor fibres and in addition a large lateralis component and a small general cutaneous bundle which joins it extra-cranially from the Gasserian ganglion and distributes to the skin of the lower part of the operculum. The two last are to be regarded as secondary additions, the former following the differentiation of the operculo-mandibular canal and the latter the backward growth of the operculum. That these general cutaneous fibres of bony fishes have not persisted from a primordial condition in which cutaneous nerves were normally present in branchial nerves is suggested by the fact that they do not emerge with the facial root, but swing back from the trigeminus. If the facialis ever possessed a general cutaneous component properly its own, in all known vertebrate types its ganglion has secondarily fused with the trigeminal general cutaneous (Gasserian) ganglion.

The pre-trematic facial is strictly typical, containing only communis fibres. In some fishes (but not in Menidia) this ramus seems to have secondarily fused after the obliteration of the spiracle, with the r. mandibularis V and to be represented in part by the chorda tympani of higher vertebrates.

The r. palatinus of the facial segment is also typical, save that it is longer than in the other nerves.

There is no general cutaneous lateral branch of the facial segment, though possibly the r. buccalis may represent such a nerve to which lateralis fibres were added and then the original general cutaneous component disappeared. For this no satisfactory evidence can be adduced. The r. oticus, however, may represent a dorsal branch. For we have seen that this nerve contains general cuta-

neous fibres. These now arise from the Gasserian ganglion and we should have to assume that their ganglion and root have secondarily fused with the trigeminal, as the profundus ganglion is supposed to have done.

The trigeminus segment has suffered still greater modification. The root contains no communis fibres, for there is no vertebrate known in which there is a pre-facial fasciculus communis. The post-trematic ramus, *i. e.*, the r. mandibularis V, has viscero-motor and general cutaneous fibres, and, as we have just seen, communis fibres belonging to the pre-trematic VII may secondarily be distributed peripherally with this ramus.

The pre-trematic ramus, or r. maxillaris, has typically only general cutaneous fibres. In some, perhaps most, fishes there are joined to these also some communis fibres from the geniculate ganglion for taste buds about the upper lip, but these are not proper trigeminal fibres.

The absence of a pre-facial fasciculus communis and communis root of the trigeminus involves the lack of a r. palatinus for this segment. Functionally this is replaced by the forward extension of the r. palatinus VII.

The r. ophthalmicus superficialis V may represent a dorsal branch of typical form, to which communis fibres are added in some types for terminal buds on the top of the head and to which the lateralis fibres of the r. ophthalmicus superficialis VII may also be joined. The r. ophthalmicus profundus might possibly represent a lateral ramus of this segment, though more probably it belonged originally to a segment lying farther cephalad and is only secondarily joined to the trigeminus segment.

III.—The Usurpation of Nerves.

From the preceding account it appears that the modifications to which the primitive segmental nerves of the head have been subjected are of two main types: in the first place by the loss of some one or more of the primary components, as the loss of the general cutaneous fibres of the IX and VII, or by the addition of components not primarily present, as in the case of the addition of special cutaneous components to the VII; and in the second place by the prolongation of nerves of one segment so that they encroach upon the area of another. This encroachment may take place either by the terminal organ of the nerve migrating secondarily into the adjacent segment and carrying its nerve with it, or the nerve may effect secondary connection with the terminal organ which belongs primarily in the adjacent segment. The occurrence of the latter case, it is true, is somewhat doubtful and is denied absolutely by some authors. Yet it would seem in the present state of our knowledge to be at least probable in some cases, as, for example, the r. intestinalis vagi, and the innervation of the skin of the head between the vagus and trigeminus by general cutaneous fibres from these nerves.

The way in which a nerve can be carried to the most distant parts of the body by a vagrant terminal organ is best illustrated by the development of the lateral lines and their nerves, as described by Wilson ('91 and '97), Ayers ('92), Mitrophanow ('93), Platt ('96) and others. The development of the so-called hypoglossus musculature from the post-otic myotomes is another illustration and Ruge ('97) suggests the same for the development of the facial musculature of the mammals, viz., that these

muscles of expression are derived from the proper musculature of the facialis segment.

It must be left to future embryological studies to determine which of these modes of encroachment has been followed by the communis nerves which emerge with the VII, IX and X nerves and spread out over the surface of the body. And in the case of the nerves which run forward from the V, VII and IX segments into the jaws and facial regions the problem is much more difficult. If the pre-trigeminal nerves ever did conform to the primitive branchiomeric type, this conformity has been so modified in all existing vertebrates as to be unrecognizable. The embryological evidence of pre-oral branchiomeres certainly needs confirmation and the persistence and metameric constancy of the somatic musculature in these segments would tend to separate them farther from the typical branchiomeres of higher forms, at least. The sensory components of these pre-oral segments, with the probable exception of the profundus, seem to have been wholly consumed in the nerves of special sense or to have degenerated altogether. The general sensory functions of these segments must, then, be supplied from the segments farther back, and we should not say that these general sensory nerves of the first segments have been *supplanted by* those of the following ones, but that the latter have pushed forward *because of* the atrophy of the proper innervation of the first segments.

Mention should be made in this connection of the supposed "vicarious relation" between the V and VII nerves developed by Pinkus ('94) from a study of the relations in the Amphibia. He calls attention to the fact that in the aquatic Amphibia the lateralis branches of the VII are highly developed and that these branches assume

progressively less importance as we ascend to the typically terrestrial Amphibia. He also assumes, though no evidence is given for it, that as the lateralis branches decrease in size the proper trigeminus branches increase and considers this to be a case of substitution of function, the general cutaneous branches compensating for the loss of the lateralis. The lateral line branches, Pinkus thinks, are the older and in higher forms have been supplanted by the general cutaneous.

This, I think, is a direct inversion of the actual relations. The general cutaneous fibres are unquestionably the older and are present throughout the fishes and Amphibians in sufficient numbers to innervate the entire cutaneous surface amply, and that, too, in cases where the lateralis system attains its maximum development. The latter system disappeared as it came, without materially affecting the general cutaneous system, but wholly in relation to the grade of organization of the corresponding sense organs.

IV.—Embryological Problems.

The clearest light upon these questions of metamerism will, I think, ultimately come from the embryological side, though so far, it must be confessed, this light has proved, in many cases, a false beacon. The reason is not far to seek, for, as has been pointed out by Cole ('98) the embryologists have not as a rule been able to follow the fate of the structures which they have discovered up to adult life and in most cases the exact anatomical structure of the adult organs of the types studied is unknown. Thus the brilliant speculations of Kupffer ('94) respecting the relation of the epi-branchial and supra-branchial sense organs to the terminal bud and lateral line systems of

nerves have thus far remained barren simply because the later development of these so-called sense organs is unknown. Again, the proper comprehension of the neural crest and its significance to metamerism must remain obscure until we learn to which of the components of the adult the cells thus derived are related. The fact that the neural crest is found in the trunk, as well as in the head, and especially Neal's account ('98, p. 238) of its relations to the vagus ganglion, suggest that it is related to the general cutaneous component only, while the special cutaneous nerves are derived from the ectodermal thickenings. But the demand in this connection is not for speculation, but for observations.

Allis, in his later paper ('97) is dominated by the conception that the sensory nerves are "split off" from the skin. This rests largely upon assumption. It is only in the case of the lateral line nerves that there is any considerable evidence for this, and even here it is by no means clear what may be the relation of the cellular strand formed in connection with the skin and the definitive fibrillar nerve. The illustrations given by Neal ('98) indicate that, whether the lateralis nerves are really split off from the skin (and this, I think, needs confirmation), it is quite evident that the general cutaneous nerves are not so derived in Acanthias. I have myself seen Neal's preparations of the r. ophthalmicus superficialis V and VII (the nerves most emphasized by Allis) and believe that such a mode of origin for the ophthalmicus superficialis V is quite out of the question.

The relations of the components as conceived by Johnston ('98) should also be criticised here. As already indicated, this author finds the centres of the acusticolateral and general cutaneous systems in the oblongata

very intimately related and he considers them but parts of a single system. The communis centre, however, is sharply separated from these, and he regards the latter system as related to entodermal structures, as distinguished from the two former which are related to ectodermal structures. These are undoubtedly related mainly, if not exclusively, to ectodermal sense organs, and it is quite possible that the acustico-lateralis was differentiated from the general cutaneous; but the communis system cannot in the existing Ichthyopsida be regarded as related even chiefly to entodermal structures. The taste buds themselves lie mainly, and probably wholly, in the region of the stomodæum, while the terminal buds of the outer skin, which are undoubtedly innervated from this system, are of course ectodermal.

It is probably true that the communis system is descended from a system of visceral nerves which was primarily related to entodermal mucous surfaces and that it has only secondarily encroached upon the ectoderm of the stomodæum and of the outer skin. These ectodermal surfaces have also retained their proper nerve supply from the trigeminus. This is illustrated by such pathological cases as that of Adolf Schmidt ('95), where the visceral sensation (taste) of the anterior two-thirds of the tongue was totally lost, but tactile sensation not so profoundly affected.

Section 13.—Conclusions.

I.—General.

In addition to this general review, a summary will be found at the close of each of the sections and to these the reader is referred for the chief anatomical and morphological findings.

The general result of the study is to demonstrate that it is possible, even in forms so highly specialized as the teleosts, to trace in serial sections the entire courses of the chief sensory and motor components of the cranial nerves and that the results of such an analysis show a striking fundamental agreement in the plan of the nervous system with the Amphibia, as worked out by Strong. This plan, in its main outlines, seems to be a palingenetic character of great constancy throughout the vertebrates. As in the Amphibia, the sensory components of the cranial nerves, to which attention has been especially directed, fall into three categories, each with its distinct terminal nuclei within the brain, roots, ganglia and peripheral branches. The latter, however, may be secondarily fused and modified in a great variety of ways, so that the peripheral nerves, as commonly named, do not usually bear a simple relation to the roots and ganglia from which they arise; much less can they be regarded as simple metameric units.

On the contrary, each of the sensory cranial systems has been, for physiological reasons, unified and concentrated in the medulla oblongata, and in consequence of this, it has come to be represented in the nerves of but few of the segments, either having been lost or not having been differentiated in the others. Thus it happens that any peripheral ramus may be composed of elements which are not only very diverse functionally, but which may have belonged primitively to different metameres.

Of these systems the general cutaneous is probably the oldest phylogenetically. It has been subjected to very slight modification in the head as compared with its arrangement in the spinal nerves, though it is represented in the V and X nerves only.

The communis system is also probably very ancient in its simple visceral form. It has, however, no pre-facial representative, and in the nerves in which it persists it has been highly modified in connection with the taste bud and terminal bud apparatus.

The acustico-lateral system seems to have been differentiated rather late and in connection primarily with the facialis segment.

The criteria of these components are primarily the central and peripheral relations of the nerves. It happens, however, that each component has certain characteristic and quite constant differences in the character of its nerve fibres which make it possible to separate them, as a rule, throughout their peripheral courses, even when several components are bound up together in a common trunk. Thus, the somatic motor fibres are always large with wide medullary sheaths and large axis cylinders, the visceral motor fibres are usually very small with very feeble myelination, the communis fibres are very minute and with still more delicate medullary sheaths, the general cutaneous fibres are of small or medium size with occasional larger fibres scattered among them, their sheaths being somewhat heavier than those of the communis fibres, and the lateralis fibres are the largest of all, having very large axis cylinders and wide sheaths which usually stain more densely than those of the somatic motor fibres.

These fibre characters, however, are not absolutely constant, but vary with the degree of development of the organs innervated. The clearest illustration of this is in the motor components. The branchial muscles, known to be of visceral origin, have acquired in the fishes the striation and large size of the fibres characteristic

of the voluntary muscles of the somatic series; and in correlation with this modification, we find that their nerve fibres, though quite certainly belonging to the viscero-motor series, are of large size, like the somatic motor nerves. Even among the somatic motor nerves it is a general rule that small muscle fibres are innervated by smaller nerve fibres than are larger muscle fibres. This is illustrated best in the eye-muscle nerves, but frequently also in the general somatic musculature. Among the sensory nerves, too, the size of the fibres seems to depend somewhat upon the state of development of the sense organ to be innervated. Thus, while the canal organs of the lateral lines are always supplied by very large fibres with wide sheaths, when the canals disappear and the organs lie exposed on the skin, as in a portion of the infra-orbital line and in the main line of the trunk, these organs are usually smaller than those in the canals, and are supplied by smaller nerve fibres. And particularly the lateralis fibres which supply the small naked organs of the " pit-lines " are always of medium or even small size, though they have the characteristic very densely stained sheaths, so that they can be easily distinguished from general cutaneous fibres of the same size. And, again, the communis fibres, though very small when distributed to visceral surfaces or taste buds on mucous surfaces, may become somewhat larger and more heavily myelinated when they distribute to large terminal buds of the outer surface of the body, so that it is sometimes impossible to distinguish them from lateralis fibres for the "pit-organs." This, however, is not always the case, for in sections of the siluroids and cyprinoids, where the terminal bud system is much more highly developed than it is in Menidia, I find these organs innervated by the very fine

fibres in the manner typical for organs of the communis system.

II.—Recapitulation of the Nerves.

The review of the components of the several nerves can best be done graphically by means of the diagrams, Figs. 8 to 12. These diagrams are composites constructed from a series of camera outlines of transections of the brain running through the entire extent of the root area of the nerve in question and are drawn with a uniform magnification. They are somewhat schematic, but they are not theoretical, as only the components which I have observed in Menidia are included. They are, of course, far from complete and other components than those figured are doubtless present in many, if not all, of these nerves. In reading the following pages these diagrams giving projections upon the transverse plane should be compared with the projections upon the sagittal plane made from the same series of sections and given in Figs. 3 to 5.

1.—*The Spinal Nerves.*

The diagram, Fig. 8, exhibits the relations as seen at the level of the fourth spinal. The large ventral root passes through the ganglion into each of the rami in the typical manner. The dorsal root is very small, much smaller, apparently, than the combined sensory components of the rami which leave the ganglion. Fibres are seen to pass from the sympathetic chain into each of the rami. The ventral ramus is the largest and contains more sensory than motor fibres. The ramus medius is rather large and contains rather more motor than sensory fibres. The two dorsal rami are both small. The cephalic one, the r. communicans, is wholly sensory, the caudal one, r. spinosus, is wholly motor. They both run bodily into the

r. lateralis accessorius dorsally, from which they distribute to the skin and adjacent muscles. The r. lateralis accessorius appears to serve as a collector for these nerves, though there is no evidence that any fibres pass in it very far from the segment in which they are related to the spinal cord.

Viscero-motor fibres are doubtless present, here as in other cases, in both the dorsal and ventral roots, though my methods have not differentiated them. Such fibres probably originate from the cells of the paracentral nucleus (*pc.n.*) lying ventro-laterally of the canalis centralis and in what might be termed the cervix of the ventral cornu. There are probably also viscero-sensory connections of some sort in the spinal cord, though what they are in the fishes remains problematical.

2.—The Vagus.

The relations of the components in the vagus nerve are shown in Fig. 9. The motor fibres all belong to the viscero-motor type and come from the nucleus ambiguus and the commissura accessoria of Mauthner. The latter probably come partly from the n. ambiguus of the opposite side and partly from the fasciculus longitudinalis dorsalis. They distribute to the pharyngeal muscles and to the m. trapezius (n. accessorious). There are probably other motor fibres of smaller calibre which distribute to the unstriated visceral musculature.

The communis root from taste buds, terminal buds and general visceral surfaces terminates in the lobus vagi. It makes up by far the largest part of the vagus.

The general cutaneous root arises from the jugular ganglion, which gives rise peripherally to the rami cutanei dorsales vagi. Centrally its fibres terminate in

the spinal V tract. This tract is drawn in Fig. 9 above the communis root, but in Fig. 4 below that root. As a matter of fact, it lies below the cephalic portion of the root, but above the caudal portion. These fibres supply the skin of the occipital region and of the dorsal part of the operculum.

The lateral line root is the most dorsal and cephalic member of the vagus complex. It terminates in the tuberculum acusticum and supplies all of the lateral line organs which are not supplied from the VII nerve.

3.—The Glossopharyngeus.

There are but two components present, as shown in Fig. 10. The motor root arises from the cephalic end of the nucleus ambiguus and is the exact counterpart of the motor root of the vagus. It runs out under the spinal V tract and distributes to the muscles of the first gill.

The communis root passes in under the root of the r. lateralis vagi and over the spinal V tract to terminate in the fasciculus communis near its entrance into the lobus vagi. These fibres distribute to the hinder surface of the first gill cleft, *i. e.*, to the first functional gill, exclusively—taste buds and general mucous surfaces—and are extended forward ventrally as a lingual nerve to the tip of the hyo-branchial apparatus. From the sensory root a small anastomosing branch runs up to join the root of the r. lateralis vagi.

4.—The Auditory Nerve.

This nerve terminates in the tuberculum acusticum and cerebellum along with the lateralis roots of the X and VII nerves. The details of its central connections were not investigated. So far as known it contains only acustico-lateralis fibres.

5.—*The Facialis.*

There are four roots and three components, as diagramed in Fig. 11. The motor root arises from the motor VII nucleus and is related in passing out to the fasciculus longitudinalis dorsalis. It runs out under the spinal V tract and the VIII root, while the other VII roots pass over these structures. It runs into the truncus hyomandibularis and supplies the mm. levator operculi, adductor operculi, adductor hyomandibularis, adductor arcus palatini and hyo-hyoideus. These are viscero-motor fibres like those from the nucleus ambiguus.

The communis root enters the brain between the two lateralis roots, arising in the geniculate ganglion and forming the whole of the pre-auditory fasciculus communis. From the geniculate ganglion fibres go out to form the whole of the r. pre-trematicus VII for the pseudobranch and the mucous lining of the mouth adjacent, and of the r. palatinus for the mucous lining and taste buds of the roof of the mouth and of the r. lateralis accessorius for special cutaneous sense organs. Other fibres enter the truncus hyomandibularis and supply the mucosa and taste buds of the lining of the mandible and lower lip; others enter the r. maxillaris of the truncus infra-orbitalis and supply taste buds of the upper lip; while still others enter the truncus supra-orbitalis.

The two lateralis roots terminate together in the tuberculum acusticum. The ventral one enters the truncus hyomandibularis and supplies the organs of the operculo-mandibular line. The dorsal one distributes its fibres to the supra- and infra-orbital trunks for the organs of the supra-orbital and infra-orbital lateral lines respectively.

6.—*The Abducens.*

The sixth nerve arises by two roots, each coming in part from the abducens nucleus and in part from the fasciculus longitudinals dorsalis. It is a pure somatic motor nerve and has no connections with any other nerve.

7.—*The Trigeminus.*

In this nerve I have found but two components (Fig. 12). The motor root is like that of the VII nerve. The configuration of the oblongata is such at this point that it emerges really dorsally of the sensory root. It enters the r. mandibularis of the infra-orbital trunk and supplies the mm. depressor operculi, levator arcus palatini, adductor mandibulæ, genio-hyoideus and intermandibularis.

The general cutaneous root, after its entrance into the brain, sends some fibres to the chief sensory trigeminal nucleus and also makes up the whole of the pre-vagal spinal V tract. From its ganglion (the Gasserian g.) fibres enter the supra-orbital trunk (r. ophthalmicus superficialis V), the infra-orbital trunk (r. maxillaris and r. mandibularis V) and two twigs (only the more cephalic one shown on Fig. 12, *t. f. 1*) run back to enter the truncus hyomandibularis for the skin of the lower part of the operculum. In addition to these, a few fibres go out with the radix longa of the ciliary ganglion which are apparently general cutaneous, rather than sympathetic and which I homologize with the r. ophthalmicus profundus trigemini.

8.—*The Trochlearis.*

This is, so far as known, a pure somatic motor nerve, arising from its nucleus in the floor of the mesocœle and doubtless also partly from the fasciculus longitudinalis

dorsalis. In its intra-cranial course it is intimately related to the origin of the r. lateralis accessorius, but no interchange of fibres could be demonstrated.

9.—The Oculomotorius.

The third nerve, like the fourth, is a pure somatic motor nerve. The origin is essentially as in the last case and the nerve peripherally has no connections with any other nerve save with the ciliary ganglion.

III.—Review of the Cranial Components.

1.—The General Cutaneous System.

Under this head I have included all nerves, exclusive of the sympathetic, which terminate free in the skin without specialized end organs. These are mainly nerves of the tactile sense, but others are also doubtless included and this system will ultimately have to be broken up into several sensory systems. In the head this system includes the two general cutaneous ganglia, the Gasserian and jugular ganglia, the root fibres from these ganglia, including the spinal V tract, and the terminal nuclei of these root fibres, viz., the chief sensory nucleus of the V nerve and the nucleus funiculi. The secondary tracts from these nuclei I have not studied exhaustively. They should also be included in a full account of the system.

The morphology of this system is perfectly simple. It corresponds to the sensory system of the dorsal horns of the spinal cord. It has suffered less modification in the head than any of the other systems, the most important being its suppression in all but the V and X cranial nerves. If it survives in any of the other nerves, it is in so altered form as to be unrecognizable. See Section 3, I; Section 5, I, 3 and VIII; Section 7.

2.—*The Acustico-lateral System.*

These nerves are related exclusively to the organs of the lateral line canals and allied sense organs, and to the similar organs of the internal ear. The system includes the ganglion of the acustic nerve, the ganglion of the r. lateralis vagi and the dorsal and ventral lateralis ganglia of the facialis, together with their root fibres and their common terminal nuclei in the tuberculum acusticum and cerebellum. Most of these root fibres terminate soon after their entrance into the brain in the tuberculum acusticum, but some turn cephalad to terminate in the cerebellum, while others turn as abruptly caudad to form a spinal VIII tract.

This system has no representative in the spinal nerves. The extreme dorso-lateral position of its terminal nucleus and of the ascending and descending root bundles suggests that this system was the last sensory system to be differentiated in the medulla oblongata. It is closely related to the general cutaneous system; nevertheless from a study of Weigert preparations of Menidia I cannot agree with Johnston ('98), who finds from a study of Golgi preparations of Acipenser that the acustico-lateral and the general cutaneous nerves belong to a single system with a common terminal centre in the oblongata. The two systems are very distinct from each other in Menidia both centrally and peripherally. It is possible that the acustico-lateral system has been derived in the phylogeny from the general cutaneous, a view which has been expressed by Cole ('97, p. 234) on embryological grounds.

See Section 2; Section 3, II; Section 5, I, 5 and X; Section 6; and Section 7.

3.—The Communis System.

This system was composed primarily of the viscero-sensory nerves, though as we actually find it in the head much has been added upon this foundation; thus, it includes not only nerves to the visceral or mucous surfaces, but to taste buds, to terminal buds of the outer surface and to the teeth. It is represented in the X, IX and VII nerves only, including all of the sensory IX, all but the lateralis portion of the sensory VII and all but the lateralis and general cutaneous portion of the sensory X. Its ganglia are the geniculate ganglion of the VII nerve, the whole of the IX ganglion and the branchio-visceral ganglia of the vagus. All of these fibres terminate in the lobus vagi—the vagus fibres directly, the others through the mediation of the fasciculus communis. There is no "lobus trigemini" or pre-vagal terminal nucleus, as in many other fishes, this being correlated with the reduction of the terminal bud system in Menidia.

The viscero-sensory system of the trunk seems to have been largly supplanted by the r. intestinalis of the vagus, yet the spinal nerves retain a vestige at least of this system. The centre in the spinal cord is uncertain, though in the higher forms Clarke's column seems to be related directly or indirectly to these fibres. In the fishes the corresponding region, the "intermediate zone" probably contains a similar centre. This is suggested by the way in which the descending tract from the lobus vagi after passing the commissura infima Halleri runs back into the intermediate zone.

See Section 3, III; Section 5; Section 7; and Section 12.

4.—The Motor Components.

The topographical relations of the motor nerves have been fully worked out. These components were not the

primary objects of study in this research and the teleosts are too highly specialized forms to reveal to the best advantage the fundamental relationships of the motor centres. Nevertheless, accepting the distinction between somatic motor and visceral motor nerves, as now commonly held by the morphologists, it appears that Menidia conforms to the usual schema given for the vertebrates; that is, the eye-muscle nerves belong to the somatic musculature and all of the other cranial motor nerves to the visceral musculature. The latter has been very highly developed in the head to form the branchial musculature. These muscles, to increase their physiological efficiency, have become striated and the nerve fibres which supply them are of large size like the other nerves for the voluntary musculature. Responding to this demand, specialized centres of origin in the oblongata have appeared for these nerves, viz., the nucleus ambiguus and the motor nuclei of the VII and V nerves, and these nuclei are related to the great longitudinal medium of muscular co-ordination, the fasciculus longitudinalis dorsalis, just like the other voluntary nerve centres of the somatic series.

The well-known relations of the motor nuclei of the several cranial nerves to this fasciculus are such as to leave no doubt that it is physiologically a very important medium of correlation of the various cranial and spinal motor centres. The fact that it is related to both the somatic and the visceral (branchio-motor) nuclei of origin makes its morphological interpretation rather perplexing. Its relations to the cranial nerve roots appear to be effected mainly, at least, through the medium of collaterals.

The findings among the motor nerves to which attention is especially directed are, in addition to the preceding points, the following:

1.—The presence in the spinal cord of two motor nuclei, the ventral horn and the paracentral nucleus. The former is a somatic centre and is probably represented in the head by the eye-muscle nerves only; the latter is probably a visceral centre, represented in the head by the viscero-motor nuclei, viz., the motor X, IX, VII and V. See Section 3, III and IV.

2.—The first spinal is a fusion of two segmental nerves. The more cephalic one (occipito-spinal nerve *b* of Fürbringer) contributes a part to the brachial plexus, the remainder supplies the post-hyal hypoglossus musculature. The pre-hyal hypoglossus musculature is wanting in the teleosts and, in correlation with this, the spino-occipital nerve *a* of most other vertebrates is reduced. See Section 4, V.

3.—The post-hyal ventral musculature is innervated by the first spinal nerve, as usual. The pre-hyal hypoglossus musculature is functionally replaced in the teleosts by a derivative of the constrictor system of the trigeminal segment of the selachians, viz., the so-called m. genio-hyoideus and the intermandibularis. These muscles in Menidia are innervated from the motor V (not motor VII, as commonly described), and can have nothing to do with the true ventral musculature. The first spinal nerve suffers a corresponding reduction. See Section 7, IV, 5, *iv*.

4.—The pharyngo-clavicularis muscles are innervated from the vagus and not from the first spinal. This differs from the accounts of some others, especially Fürbringer, and will necessitate some modifications in that author's scheme of the relations of somatic and visceral muscles in the vagus region of teleosts. See Section 5, VII, 5, *v*.

5.—There is a branch of the vagus for the m. trapezius, *i. e.*, a true spinal accessory nerve, in Menidia. Section 5, IX.

IV.—Special Results.

Among the more specific results to which attention is especially invited are the following:

1.—The fasciculus communis and associated structures of the Ichthyopsida are in a general way homologous with the fasciculus solitarius and its associated structures of the Amniota, though the homology is not exact.

2.—The innervation of the pseudobranch from the facial nerve supports Maurer's later view that the teleostean pseudobranch represents a spiracular demibranch or mandibular gill. This organ is very highly developed in Menidia and has invaded the post-spiracular or hyoidean region of the pharynx so that the pre-trematic ramus of the IX nerve has entirely disappeared.

3.—The nerve to the pseudobranch represents a pre-trematic ramus of the facialis and supplies the adjacent pharyngeal mucosa. This nerve coexists in some fishes with the chorda tympani, or pre-spiracular extension of the communis component for the hyoid and mandibular arches. The chorda is absent in Menidia and the post-trematic r. mandibularis internus VII of fishes cannot be homologized with it, though in higher forms it is possible that the two nerves fuse into a common trunk.

4.—The ophthalmicus profundus is apparently represented by a vestigeal bundle of general cutaneous fibres which run out from the Gasserian ganglion with the radix longa of the ciliary ganglion. Having reached the latter ganglion, they can no longer be traced.

5.—The sensory epithelium of the olfactory organ

exhibits the "olfactory buds" of Blaue, which are so general among the teleosts. The development, however, shows that these are not survivals of a more primitive condition, but that they are late and secondary acquisitions. Blaue's supposition that they are related to the lateral line organs or terminal buds is impossible for several reasons.

6.—I would reiterate the position taken by most of the recent students of nerves, that the morphological value of a given nerve is to be determined primarily by its terminal relations, *i. e.*, its central nucleus and its peripheral end-organ. These appear to be very constant, while its intermediate course may be modified by so many cenogenetic factors as to be of relatively small value in determining the homologies.

7.—Finally, I would urge that the significance of the sensory components of the cranial nerves for metamerism has been greatly misunderstood. The consequences following the attempt to compare all sensory cranial nerves directly with dorsal spinal roots and to apply Bell's law in its simplest form to the cranial nerves have been so disastrous to sound morphology that the tendency among the most recent writers seems to be to deny the metameric value of the sensory cranial roots altogether and to confine attention to the motor roots. This is also too extreme. The problems of metamerism in the case of the sensory roots are vastly more complicated than in the case of the motor; yet I do not believe that they are insoluble. Some suggestions as to the lines along which I think the solution is to be sought are given in the preceding section.

December 31, 1898.

THE LITERATURE CITED.

ALCOCK, R.

'98. The Peripheral Distribution of the Cranial Nerves of Ammocœtes. *Journal of Anatomy and Physiol.*, XXXIII, N. S., XIII, 1.

ALLIS, E. P., JR.

'89. The Anatomy and Development of the Lateral Line System in Amia calva. *Journ. Morph.*, II, 3.

'95. The Cranial Muscles and Cranial and First Spinal Nerves in Amia calva. (Preliminary). *Journ. Morph.*, XI, 2.

'97. The Cranial Muscles and Cranial and First Spinal Nerves in Amia calva. *Journ. Morph.*, XII, 3.

'98. The Homologies of the Occipital and First Spinal Nerves of Amia and Teleosts. *Zoolog. Bulletin*, II, 2.

AMABILINO, R.

'98. Sui rapporti del ganglio genicolato con la corda del timpano e col facciale. Richerche sperimentali. *Il Pisani*, XIX, 1–2.

APOLANT, HUGO

'96. Ueber die Beziehung des Nervus oculomotorius zum Ganglion ciliare. *Arch. mik. Anat.*, XLVII, 4.

AYERS, HOWARD.

'92. Vertebrate Cephalogenesis. II. A Contribution to the Morphology of the Vertebrate Ear, with a Reconsideration of its Functions. *Journ. Morph.*, VI, 1.

BARKER, LEWELLYS F.

'97. On Certain Changes in the Cells of the Ventral Horns and of the Nucleus Dorsalis (Clarkii) in Epidemic Cerebro-Spinal Meningitis. *Brit. Med Jour.*, No. 1930.

BATTEN, F. E.

'97. The Muscle Spindle under Pathological Conditions. *Brain*, LXXVII–LXXVIII.

BAUDELOT, E.

'83. Récherches sur le système nerveux des poissons. *Paris.*

BEARD, JOHN.

'85. The System of Branchial Sense Organs and their Associated Ganglia in Ichthyopsida. A Contribution to the Ancestral History of Vertebrates. *Q. J. M. S.*, XXVI, N. S.

Blaue, Julius.

'84. Untersuchungen über den Bau der Nasenschleimhaut bei Fischen und Amphibien, namentlich über Endknospen als Endapparate des Nervus olfactorius. *Arch. f. Anat. und Phys., Anat. Abt.*

Bonnier, Pierre.

'95. Rapports entre l'appareil ampullaire de l'oreille interne et les centres oculo-moteurs. *Rev. Neurol.*, III, 23.

Böttiger, A.

'90. Beitrag zur Lehre von den chronischen progressiven Augenmuskellähmungen und zur feineren Hirn-anatomie. *Arch. f. Psychiatrie*, XXI.

Bunker, F. S.

'97. On the Structure of the Sense Organs of the Lateral Line of Ameurius nebulosus LeS. *Anat. Anz.*, XIII, 8–9.

Cajal, S. Ramón y.

'96. Beitrag zum Studium der Medulla oblongata, des Kleinhirns und des Ursprungs der Gehirnnerven. Trans. by Bresler. *Leipzig, J. A. Barth.*

Clapp, Cornelia M.

'99. The Lateral Line System of Batrachus tau. *Journ. of Morphology*, XV, 2. [I am permitted by the kindness of the author to refer to the proof sheets of this work in advance of its publication].

Cole, Frank J.

'96. On the Sensory and Ampullary Canals of Chimæra. *Anat. Anz.*, XII, p. 172.

'96 a. On the Cranial Nerves of Chimæra monstrosa (Linn.), with a Discussion of the Lateral Line System and of the Morphology of the Chorda Tympani. *Trans. Roy. Soc. Edinburgh*, XXXVIII, Part III (No. 19).

'98. Reflections on the Cranial Nerves and Sense Organs of Fishes. *Trans. Liverpool Biological Soc.*, XII.

'98 a. Observations on the Structure and Morphology of the Cranial Nerves and Lateral Sense Organs of Fishes; with Special Reference to the Genus Gadus. *Trans. Linn. Soc. London*, 2 Ser., Vol. VII, Part 5.

Collinge, Walter E.

'94. The Sensory Canal System of Fishes. Part I. Ganoidei. *Q. J. M. S.*, N. S., XXXVI.

'95. On the Sensory Canal System of Fishes. A. Physostomi. *Proc. Zool. Soc. London*, Apr., 1895.

'95a. Some Researches upon the Sensory Canal System of Ganoids. *Proc. Birmingham Nat. Hist. and Philos. Soc.*, Vol. X.

DEYL.

'95. Zur vergleichenden Anatomie des Sehnerven. *Prague.*

DIXON, A. FRANCIS.

'96. On the Development of the Branches of the Fifth Cranial Nerve in Man. *Trans. Roy. Dublin Soc.*, Ser. 2, VI.

EDINGER, L.

'96. Vorlesungen über den Bau der Nervösen Centralorgane des Menschen und der Thiere. 5 Ed., *Leipzig.*

EMERY, CARLO.

'80. Le specie del genere Fierasfer nel Golfo di Napoli e regioni limitrofe. *Fauna und Flora des Golfes von Neapel. II Monographie.*

EWART, J. C.

'89. On the Cranial Nerves of the Elasmobranch Fishes. Prelim. Com. *Proc. Roy. Soc.*, XLV.

'90. The Cranial Nerves of the Torpedo. Prelim. Note. *Proc. Roy. Soc.*, XLVII.

'92. Supplementary Note on the Cranial Nerves of Elasmobranchs. *Edinburgh.*

'93. The Lateral Line Sense Organs of Elasmobranchs. I. The Sensory Canals of Læmargus. *Trans. Roy. Soc. Edinburgh*, XXXVII, 1 Pt., Nos. 5–6.

EWART, J. C. AND COLE, F. J.

'95. On the Dorsal Branches of the Cranial and Spinal Nerves of Elasmobranchs. *Proc. Roy. Soc. Edinburgh*, XX.

EWART, J. C. AND MITCHELL, J. C.

'93. The Lateral Sense Organs of Elasmobranchs. II. The Sensory Canals of the Common Skate (Raja batis). *Trans. Roy. Soc. Edinburgh*, XXXVII, Pt. 1, Nos. 5–6.

FOREL, AUG.

'91. Ueber das Verhältniss der experimentellen Atrophie und Degenerationsmethode zur Anatomie und Histologie des Centralnervensystems. Ursprung des IX, X und XII Hirnnerven. *Festschr. f. Nägeli und Kölliker, Zurich.*

Froriep, A.

'87. Ueber das Homologon der Chorda tympani bei niederen Wirbelthieren. *Anat. Anz.*, II, p. 486.

Fürbringer, Max.

'97. Ueber die spino-occipitalen Nerven der Selachier und Holocephalen und ihre vergleichenden Morphologie. *Festschr. f. Gegenbaur*, Bd. 3.

Garman, S.

'88. On the Lateral Canal System of Selachia and Holocephala. *Bul. Mus. Comp. Zool.*, XVII, 2.

Gaskell, W. H.

'86. On the Structure, Distribution and Function of the Nerves which Innervate the Visceral and Vascular Systems. *Jour. of Physiol.*, VII.

'89. On the Relation between the Structure, Function, Distribution and Origin of the Cranial Nerves, together with a Theory of the Origin of the Nervous System of Vertebrata. *Journ. of Physiol.*, X.

Gehuchten, A. van.

'95. La moelle épinière de la truite (Trutta fario). *La Cellule*, XI, 1.

'97. Anatomie du système nerveux de l'homme. 2. Ed., *Louvain.*

Goronowitsch, N.

'88. Das Gehirn und die Cranialnerven von Acipenser ruthenus. *Morph. Jahrb.*, XIII.

'96. Der Trigeminofacialis-Complex von Lota vulgaris. *Festschr. f. Gegenbaur*, Bd. III.

Guitel, F.

'91. Recherches sur la ligne latérale de la baudroie (Lophius piscatorius). *Arch. Zool. Expér.*, 2 Ser., IX.

'91a. Recherches sur les boutons nerveux bucco-pharyngiens de la baudroie (Lophius piscatorius). *Arch. Zool. Expér.*, 2 Ser., IX.

Haller, B.

'96. Der Ursprung der Vagusgruppe bei den Teleostiern. *Festschr. f. Gegenbaur*, Bd. III.

Harrison, R. G.

'95. Die Entwickelung der unpaaren und paarigen Flossen der Teleostier. *Arch. f. mik. Anat.*, XLVI.

HERRICK, C. JUDSON.

'94. The Cranial Nerves of Amblystoma punctatum. *Journ. Comp. Neurol.*, IV.

'97. The Cranial Nerve Components of Teleosts. *Anat. Anz.*, XIII, 16.

'98. Report upon a Series of Experiments with the Weigert Methods with Special Reference for Use in Lower Brain Morphology. *State Hospitals Bull.*, Oct., '97; issued May, '98. Full auto-abstract in *Journ. Comp. Neurol.*, July, '98.

'98 a. The Cranial Nerves of Bony Fishes. *Jour. Comp. Neurol.*, VIII, 3.

HERRICK, C. L.

'91. Studies on the Brains of some American Fresh-water Fishes. *Jour. Comp. Neurol.*, I, p. 228–245.

HERTWIG, O.

'92. Text-book of the Embryology of Man and Mammals. Translated from the third German edition by E. L. Mark. *London.*

HOLM, JOHN F.

'94. The Development of the Olfactory Organ in the Teleostei. *Morph. Jahrb.*, XXI, 4.

HUBER, G. CARL.

'97. Lectures on the Sympathetic Nervous System. *Journ. Comp. Neurol.*, VII, 2.

HUBER, G. CARL AND DEWITT, LYDIA M. A.

'98. A Contribution on the Motor Nerve-Endings and on the Nerve-Endings in the Muscle Spindles. *Journ. Comp. Neurol.*, VII, 3–4.

JACKSON, W. H. AND CLARKE, W. B.

'76. The Brain and Cranial Nerves of Echinorhinus spinosus. *Journ. Anat. and Physiol.*, X.

JENDRÁSSIK, ERNST.

'96. Allgemeine Betrachtungen über das Wesen und die Function des vegetativen Nervensystems. *Arch. f. path. Anat.*, CXLV, 3.

JOHNSTON, J. B.

'98. Hind Brain and Cranial Nerves of Acipenser. *Anat. Anz.*, XIV, 22–23.

JULIN, C.

'87. Recherches sur l'appareil vasculaire et le système nerveux peripherique de l'Ammocœtes (Petromyzon planeri). *Archives de Biol.*, VII.

KINGSBURY, B. F.

'95. On the Brain of Necturus maculatus. *Journ. Comp. Neurol.*, V, 3.

'97. The Structure and Morphology of the Oblongata in Fishes. *Journ. Comp. Neurol.*, VII, 1.

v. KÖLLIKER, A.

'94. Ueber die feinere Anatomie und die physiologische Bedeutung des sympathischen Nervensystems. *Ges. Deutscher Naturforscher und Aerzte*, Verh. 1894, Allgemeiner Theil.

'96. Handbuch der Gewebelehre des Menschen. II Bd., 6. Ed., *Leipzig*.

v. KUPFFER, C.

'91. The Development of the Cranial Nerves of Vertebrates. Trans. by O. S. Strong. *Journ. Comp. Neurol.*, I.

'94. Die Entwicklung des Kopfes von Ammocœtes planeri. Studien zur vergl. Entwg. des Kopfes d. Kranioten. Heft II. *Munich.*

'96. Entwickelungsgeschichte des Kopfes. *Merkel und Bonnet's Ergenbnisse* for 1895, pp. 562–618.

LANGLEY, J. N.

'98. On the Union of Cranial Autonomic (Visceral) Fibres with the Nerve Cells of the Superior Cervical Ganglion. *Jour. of Physiol.*, XXIII, 3.

LANGLEY, J. N. AND ANDERSON.

'92. The Action of Nicotin on the Ciliary Ganglion and on the Endings of the Third Cranial Nerve. *Jour. of Physiol.*, XIII.

LEE, F. S.

'98. The Functions of the Ear and the Lateral Line in Fishes. *Am. Journ. of Physiol.*, I.

v. LENHOSSÉK, M.

'94. Das Ganglion geniculi Nervi facialis und seine Verbindungen. In *Beiträge zur Histologie des Nervensystems und der Sinnesorgane. Wiesbaden.*

LEYDIG, F.

'94. Integument und Hautsinnesorgane der Knochenfische. Weitere Beiträge. *Zool. Jhrb. Abt. f. Anat. u. Ontogen.*, VIII, 1.

MADRID-MORENO, J.

'86. Ueber die morphologische Bedeutung der Endknospen in der Riechschleimhaut der Knochenfische. Bericht von C. Emery. *Biol. Cent.*, VI.

MARSHALL, A. M. AND SPENCER, W. B.

'81. Observations on the Cranial Nerves of Scyllium. Part I. The Pre-Auditory Nerves. *Q. J. M. S.*, XXI.

MAURER, F.

'84. Ein Beitrag zur Kenntnis der Pseudobranchien der Knochenfische. *Morph. Jhrb.*, IX.

'88. Die Kiemen und ihre Gefässe bei anuren und urodelen Amphibien und die Umbildung der beiden ersten Arterienbogen bei Telieostiern. *Morph. Jhrb.*, XIV.

MAYSER, P.

'81. Vergleichend-anatomische Studien über das Gehirn der Knochenfische mit besonderer Berücksichtigung der Cyprinoiden. *Zeits. f. wiss. Zool.*, XXXVI, 2.

MCMURRICH, J. PLAYFAIR.

'84. The Myology of Amiurus catus (L) Gill. *Proc. Canadian Institute*, N. S., II.

'85. The Cranial Muscles of Amia calva L., with a Consideration of the Relations of the Post-occipital and Hypoglossal Nerves in the Various Vertebrate Groups. *Studies from the Biol. Lab., Johns Hopkins Univ.*, III.

MERKEL, FR.

'80. Ueber die Endigungen die sensiblen Nerven in der Haut der Wirbelthiere. *Rostock.*

MICHEL.

'94. *Verhandlung des 8. Intern. Ophthalmol. Congr. in Edinburgh.*

MINOT, C. S.

'92. Human Embryology. *New York.*

MITROPHANOW, P.

'93. Etude embryogènique sur Sélaciens. *Arch. de Zool. Exp. et Gen.*, 3 Ser., I.

Morrill, A. D.

'95. The Pectoral Appendages of Prionotus and their Innervation. *Journ. Morphology*, XI.

Müller, Friedrich N.

'97. Ueber die Entwicklung und morphologische Bedeutung der "Pseudobranchie" und ihre Umgebung bei Lepidosteus osseus. *Arch. f. mik. Anat.*, XLIX, 3.

Neal, H. V.

'97. The Development of the Hypoglossus Musculature in Petromyzon and Squalus. *Anat., Anz.*, XIII, 17.

'98. The Segmentation of the Nervous System in Squalus acanthias. A Contribution to the Morphology of the Vertebrate Head. *Bul. Mus. Comp. Zool.*, XXXI, 7.

'98a. The Problem of the Vertebrate Head. *Journ. Comp. Neurol.*, VIII, 3.

Niedzvietzky, W.

'97. Zur Frage über Veränderungen im Nervensystem und in inneren Organen nach der Resection des N. vagus und des N. splanchnicus. *Bull. Soc. Imp. der Nat. de Moscou*, Année, 1896, No. 3.

Onuf, B. and Collins, Joseph.

'98. Experimental Researches on the Localization of the Sympathetic Nerve in the Spinal Cord and Brain, and Contributions to its Physiology. Abstract. *Journ. Nerv. and Ment. Dis.*, XXV, 9.

Osborn, H. F

'88. A Contribution to the Internal Structure of the Amphibian Brain. *Journ. Morph.*, II, 1.

Owen, R.

'66. On the Anatomy of Vertebrates. *London.*

Penzo.

'93. Ueber das Ganglion geniculi und die mit demselben zusammenhängenden Nerven. *Anat. Anz.*, 1893, p. 738.

Pinkus, F.

'94. Die Hirnnerven des Protopterus annectens. *Schwalbe's Morph. Arbeiten*, IV, 2.

Platt, Julia B.

'91. A Contribution to the Morphology of the Vertebrate Head, Based on a Study of Acanthias vulgaris. *Journ. Morph.*, V.

'96. Ontogenetic Differentiation of the Ectoderm in Necturus. Study II. On the Development of the Peripheral Nervous System. *Q. J. M. S.*, XXXVIII, 4.

Pollard, H. B.
'91. Lateral Line of Siluroids. *Zool. Jhrb.*, V, 2.
'95. The Oral Cirri of Siluroids and the Origin of the Head in Vertebrates. *Zool. Jhrb.*, VIII, 3.

Retzius, G.
'81. Das Gehörorgan der Wirbelthiere. *Stockholm.*
'94. Ueber das Ganglion ciliare. *Anat. Anz.*, IX.
'94 a. Ganglion ciliare. *Biol. Unters.*, N. F., VI, 2–3.

Roller, C.
'81. Der centraler Verlauf des N. glossopharyngeus. *Arch. f. mik. Anat.*, XIX.

Ruge, Georg.
'97. Ueber das peripherische Gebiet des Nervus facialis bei Wirbelthieren. *Festschr. f. Gegenbaur*, Bd. III.

Sanders, A.
'79. Contribution to the Anatomy of the Central Nervous System in Vertebrate Animals. Part I. Ichthyopsida—Teleostei. *Phil. Trans.*, Pt. II, No. 87.

Schäfer, E. A.
'95. The Spinal Cord and Brain. Vol. III, Part I of Quain's Anatomy, tenth edition. *London.*
'97. Organs of the Senses. Vol. III, Part III of Quain's Anatomy, tenth edition. *London.*

Schmidt, Adolf.
'95. Ein Fall vollständiger isolirter Trigeminuslähmung nebst Bemerkungen über den Verlauf der Geschmacksfasern der Chorda tympani und über trophische Störungen. *Dtsch. Z. Nervenhk.*, VI, 5–6.

Schulze, F. E.
'70. Ueber die Sinnesorgane der Seitenlinie bei Fischen und Amphibien. *Arch. f. mik. Anat.*, VI.

Schwalbe, G.
'79. Das Ganglion oculomotorii. Ein Beitrag zur vergleichenden Anatomie der Kopfnerven. *Jen. Zeits.*, XIII, 2.

Shore, Thos. W.
'88. The Morphology of the Vagus Nerve. *Journ. Anat. and Physiol.*, XXII.
'89. On the Minute Anatomy of the Vagus Nerve in Selachians, with Remarks on the Segmental Value of the Cranial Nerves. *Journ. Anat. and Physiol.*, XXIII.

Stannius, H.
'49. Das peripherische Nervensystem der Fische, anatomisch und physiologisch untersucht. *Rostock.*

Strong, O. S.

'90. The Structure and Homologies of the Cranial Nerves of the Amphibia as Determined by their Peripheral Distribution and Internal Origin. *Zool. Anz.*, No. 348.

'92. The Structure and Homologies of the Cranial Nerves of the Amphibia as Determined by their Peripheral Distribution and Internal Origin. *Anat. Anz.* VII, 15.

'95. The Cranial Nerves of the Amphibia. A Contribution to the Morphology of the Vertebrate Nervous System. *Journ. Morph.*, X, 1.

'98. Review of Johnston on the Cranial Nerves of the Sturgeon. *Journ. Comp. Neurol.*, VIII, 3.

Studnicka, F. K.

'96. Untersuchungen über den Bau des Sehnerven der Wirbeltiere. *Jen. Zeits.*, XXXI.

Thane, G. D.

'95. The Nerves. Vol. III, Part II of Quain's Anatomy, tenth edition. *London.*

Vetter, B.

'74. Untersuchungen zur vergleichenden Anatomie der Kiemen- und Kiefermusculatur der Fische. *Jen. Zeits.*, VII.

'78. do. Part II. *Jen. Zeits.*, XII.

Vogt and Jung.

'94. Traité d'anatomie compareé pratique. *Paris.*

Wiedershiem, R.

'93. Grundriss der vergleichenden Anatomie der Wirbeltiere. 3 Ed. *Jena.*

Wilson, H. V.

'91. The Embryology of the Sea Bass (Serranus atrarius). *Bull. U. S. Fish Com.*, Vol. IX, for 1889, issued 1891. Washington, D. C.

Wilson, H. V. and Mattocks, J. E.

'97. The Lateral Sensory Anlage in the Salmon. *Anat. Anz.*, XIII, 24.

Wright, R. Ramsay.

'84. On the Skin and Cutaneous Sense Organs of Amiurus. *Proc. Canadian Institute*, N. S., II.

'84a. On the Nervous System and Sense Organs of Amiurus. *Proc. Canadian Institute*, N. S., II.

'85. On the Hyomandibular Clefts and Pseudobranchs of Lepidosteus and Amia. *Journ. Anat. and Physiol.*, XIX.

DESCRIPTION OF THE FIGURES.

All of the figures of Menidia, viz., all except figs. 6, 14 and 15, are based upon a single series of transections of Menidia gracilis. The findings, however, were controlled by many other series prepared by the same and other methods. The serial numbers of the sections are indicated upon the scales above and below the plotted reconstructions and all drawings of transections are given their appropriate serial number. These serial section numbers are also used frequently throughout the text as a convenient means of referring to positions on the plots. The plots are accurately drawn to scale on the basis of a series of camera lucida outlines of representative sections.

Reference Letters.

a. to *i.*—naked cutaneous sense organs about the nasal apertures.

a. c.—anterior semicircular canal.

a. l.—anastomosing root of the r. lateralis vagi derived from the n. IX.

amp. ex.—external ampulla.

ART.—articular bone.

a. r. VII., *a. r. VII. 1.* and *a. r. VII. 2.*—anastomosing rami from the vagus to the r. recurrens VII to form the r. lateralis accessorius.

b. c. and *b. c. 1.* to *b. c. 5.*—the five branchial clefts.

br. g. X.—the ganglia of the four branchial rami of the vagus, including the visceral ganglia.

b. v.—blood vessel.

cb.—cerebellum.

cb. cr.—cerebellar crest.

cb. VIII.—cerebellar VIII, root fibres from VIII nerve to cerebellum of same side.

CB3.—the third ceratobranchial bone.

c. d.—dorsal cornu of spinal cord.

cil. b.—ramus ciliaris brevis.

cil. g.—ciliary ganglion.

cil. l.—ramus ciliaris longus.

com. ac. M.—commissura accessoria Mauthneri.

com. inf.—commissura infima Halleri.

com. IX.—communis (sensory) root of the glossopharyngeus.

com. mx.—the communis element for the r. maxillaris V.

com. oph. sup.—the communis element for the r. ophthalmicus superficialis.

com. VII.—communis root of the facialis.

com. X.—communis root of the vagus.

co. 1.—fibres from the second branch of the supra-orbital trunk for the cornea.

co 2.—do. from the fifth supra-orbital branch.

cr.—cranial wall.

cut. V.—the sensory (general cutaneous) component of the trigeminus.

cut. X.—fibres from the spinal V tract to the cutaneous branches of the vagus.

cut. X. 2 and *cut. X. 3.*—rami cutanei dorsales vagi.

cut. 1.—cutaneous fibres from the first spinal to the region behind the cleithrum.

c. v.—ventral cornu of spinal cord.

D.—dentary bone.

d. b.—first dorsal root of first spinal nerve (*b* of Fürbringer).

d. c.—second dorsal root of first spinal nerve (*c* of Fürbringer).

dep.—nerves for the depressor muscle of the pectoral fin.

d. lat. VII.—dorsal lateralis root of the facialis.

d. l. g. VII.—dorsal lateral line ganglion of the facialis.

d. V.—deep root of the V nerve, containing fibres from the motor V nucleus and for the "chief sensory nucleus" of the V nerve.

d. 2. to *d. 4.*—dorsal roots of second to fourth spinal nerves.

EB4.—the fourth epibranchial bone.

e. c.—external semicircular canal.

ESC.—extra-scapular bone.

f. c.—fasciculus communis.

f. d. 2.—nerves from second spinal for skin of dorsal part of pectoral fin.

f. l. d.—fasciculus longitudinalis dorsalis.

f. m. 3.—nerve from third spinal for skin of middle part of pectoral fin.

FR.—frontal bone.

f. v. 3.—nerve from third spinal for skin of ventral part of pectoral fin.

G.—the caudal extra-cranial tip of the Gasserian ganglion, giving rise to the second anastomosing nerve, *t. f. 2.*

Gas. g.—Gasserian ganglion.

g. cœl.—ganglion cœliacum.

gen. g. VII—geniculate ganglion of the facialis.

g. IX.—ganglion of the glossopharyngeus.

g. lat. X.– ganglion of r. lateralis vagi.

g. X. 1. to *g. X. 3.*—the ganglia of the first to third trunci branchiales vagi.

g. X. 4+5.—the common ganglion of the fourth truncus branchialis vagi and the r. intestinalis vagi.

HM.—hyomandibular bone.

hy.—ramus hyoideus facialis.

hy. 1. to *hy. 5.*—branches of the r. hyoideus VII.

IH.—interhyal bone.

III.—the n. oculomotorius.

io. 1. to *io. 20.*—branches of the infra-orbital trunk.

i. p. 1 to *i. p. 4.*—pores of the infra-orbital lateral line.

is. m. b.—dorsal ramus from first spinal nerve *b* to interspinal muscles.

IV.—the n. trochlearis.

IX.—the n. glossopharyngeus.

IX-f. l. d.—fibres from the motor IX root to the fasciculus longitudinalis dorsalis.

IX-l. X.—communicating root IX to r. lateralis vagi.

jug. g.—the general cutaneous ganglion of the vagus, jugular ganglion of Shore and Strong.

k.—common communis root from geniculate ganglion for rr. maxillaris and ophthalmicus superficialis.

ki.—the head kidney.

l.—lateral reticular area of spinal cord.

LA.—lachrymal bone.

lev.—nerves for the levator muscles of the pectoral fin.

l. g. X.—ganglion of the r. lateralis vagi.

lob. inf.—lobi inferiores.

lob. X.—lobus vagi.

m. ad. a. p.—branch of r. opercularis profundus VII for m. adductor arcus palatini.

m. ad. arc. pal.—m. adductor arcus palatini.

m. ad. hy.—branch of r. opercularis profundus for m. adductor hyomandibularis.

m. ad. man.—m. adductor mandibulæ.

m. ad. op.—branch of r. opercularis profundus VII for m. adductor operculi.

m. a. m.—branches of the infra-orbital trunk (r. mandibularis V) for the m. adductor mandibulæ.

man. c.—naked organ, representing a mandibular commissure ("pit-line") on the lower lip.

man. ext. VII.—ramus mandibularis externus facialis.

man. V.—ramus mandibularis V.

man. VII—ramus mandibularis VII.

m. b. o. s.—branch of second branchial trunk for m. branchialis obliquus superior.

m. dil. o.—m. dilator operculi.

m. d. op.—branch of infra-orbital trunk for m. depressor operculi.

men.—meninges.

m. ghy.—branch of the r. mandibularis V, which, after anastomosing with the r. mandibularis VII, supplies the m. geniohyoideus.

m. im.—branch of r. mandibularis V for the m. intermandibularis.

m. l. a. p.—branch of infra-orbital trunk for m. levator arcus palatini.

m. lev. op.—branch of r. opercularis profundus VII for m. levator operculi.
m. lev. p.—m. levator arcus palatini.
m. l. op.—m. levator operculi.
m. n. V.—motor nucleus of the trigeminus.
m. n. VI—motor nucleus of the abducens.
m. n. VII—motor nucleus of the facialis.
mn. 1. to *mn. 5.*—organs of the mandibular canal.
m. o. d.—branch of third branchial n. for m. obliquus dorsalis.
m. o. d. p.—branch of fourth truncus branchialis vagi for m. obliquus dorsalis posterior.
mot. IX.—motor root of the glossopharyngeus.
mot. V.—motor root of the trigeminus.
mot. VII.—motor root of the facialis.
mot. X.—motor root of the vagus.
m. p. c. i.—the m. pharyngo-clavicularis internus.
m. ph. c. e.—branch of vagus for m. pharyngo-clavicularis externus.
m. ph. c. i.—branch of vagus for m. pharyngo-clavicularis internus.
m. ph. t.—branch of vagus for m. pharyngeus transversus.
m. p. l.—mandibular pit-line.
m. p. 1. to *m. p. 4.*—pores of the mandibular canal.
m. r. d.—branch of vagus for m. retractor arcus branchii dorsalis.
m. trap.—trapezius muscle (m. protractor scapulæ).
m. tr. d.—branch of vagus for m. transversus dorsalis.
m. VII. 1. to *m. VII. 12.*—branches of the r. mandibularis VII.
mx. V.—ramus maxillaris V.
m. 1. to *m. 3.*—organs of the main lateral line canal of the head.
m. 4.—organ of the occipital commissure.
NA.—nasal bone.
n. a.—nucleus ambiguus.
n. a. a.—anterior nasal aperture.
n. a. p.—posterior nasal aperture.
n. fn.—nucleus funiculi.
n. I.—the olfactory nerve.
n. II.—the optic nerve.
n. III.—the oculomotor nerve.
oc. c.—occipital commissure of lateral line system.
o. i.—m. obliquus inferior.
o. IX.—apparent (superficial) origin of the IX nerve.
o. i. 1 to *o. i. 15.*—organs of the infra-orbital lateral line.
o. l.—optic lobe.
o. m. VII.—apparent origin of the motor VII nerve.
OP.—opercular bone.
oph.—ramus ophthalmicus trigemini.
op. p. VII.—ramus opercularis profundus facialis.
op. p. 1. to *op. p. 6.*—pores of the opercular canal.

o. pr.—the ramus ophthalmicus profundus.

o. pr. r.—fibres from Gasserian ganglion to lateral lobe of the first sympathetic ganglion, supposed to enter the r. oph. profundus.

op. s. VII.—ramus opercularis superficialis of the truncus hyomandibularis.

op. s. VII. 1. to *op. s. VII. 5.*—first to fifth branches of r. opercularis superficialis VII.

op. X. 1. to *op. X. 4.*—branches of the ramus opercularis vagi.

op. 1. to *op. 7.*—organs of the opercular canal.

o. r. l.—apparent origin of r. lateralis vagi.

o. s.—m. obliquus superior.

o. s. 1. to *o. s. 6.*—organs of the supra-orbital canal.

o. V.—apparent origin of the V nerve.

o. VI.—apparent origin of the VI nerve.

o. X.—apparent origin of the vagus nerve.

o. 1. to *o. 4.*—organs of the ventral opercular pit-line.

o. 5.—similar organ on the dorsal edge of the operculum.

o. 6.—similar organ on the cephalic edge of the operculum.

pal.—the ramus palatinus facialis.

pb.—pseudobranch.

p. c.—posterior semicircular canal.

pc. n.—paracentral nucleus.

ph. d.—branch of the vagus for the most caudal superior pharyngeal teeth.

ph. IX.—ramus pharyngeus IX.

ph. v.—branches of the vagus for the inferior pharyngeal teeth.

ph. X. and *ph. X. 1.* to *ph. X. 3.*—pharyngeal rami of first to third branchial trunks.

PO.—most dorsal post-orbital bone.

POP.—preopercular bone.

post. and *post. 1.* to *post 4.*—post-trematic rami of the first to fourth branchial trunks of the vagus.

post. 2. d. and *post. 2. v.*—dorsal and ventral ramuli of the second post-trematic ramus.

pre. and *pre. 1.* to *pre. 2.*—pre-trematic rami of the first to fourth branchial trunks of the vagus.

PRO.—prootic bone.

PS.—parasphenoid bone.

r. a. a.—ramulus acusticus ampullæ anterioris.

r. a. e.—ramulus acusticus ampullæ externæ.

r. a. p.—ramulus acusticus ampullæ posterioris.

r. b.—m. retractor bulbi.

r. buc.—ramus buccalis facialis.

r. car.—ramus cardiacus vagi.

r. cerv.—ramus cervicalis, Fürbringer; "hypoglossus" of authors.

r. com.—ramus communicans, or sensory portion of dorsal branch of spinal nerves.

r. com. b.—ramus communicans between first spinal nerve *b* and the r. lateralis accessorius.

r. com. 2.—do. between second spinal nerve and r. lateralis accessorius.

r. com. 3.—do. do. third spinal nerve do.

r. com. 4.—do. do. fourth spinal nerve do.

r. cut. dors. X.—ramus cutaneous dorsalis vagi.

r. e.—m. rectus externus.

r. é.—fine fibred slip of m. rectus externus.

rec. 1. to *rec. 3.*—roots of the ramus lateralis accessorius (recurrent roots of the facial).

r. if.—m. rectus inferior.

r. intest. X.—ramus intestinalis vagi.

r. it.—m. rectus internus.

r. *IX.*—root of the glossopharyngeus.

r. l.—ramulus acusticus lagenæ.

r. lat. ac.—ramus lateralis accessorius.

r. lat. X.—ramus lateralis vagi.

r. l. 1. to *r. l. 4.*—the first four twigs of the r. lateralis vagi.

r. m.—ramus medius of spinal nerves.

r. m. b.—ramus medius of first spinal nerve *b*.

r. m. c.—ramus medius of first spinal nerve *c*.

r. m. 2. to *r. m. 4.*—ramus medius of second to fourth spinal nerves.

r. n.—ramulus acusticus neglectus.

r. oes.—œsophageal rami of the vagus.

r. oph. sup. V.—ramus ophthalmicus superficialis trigemini.

r. oph. sup. VII.—ramus ophthalmicus superficialis facialis.

r. op. V.—ramus opercularis trigemini.

r. op. X.—ramus opercularis vagi.

r. ot.—ramus oticus.

r. ot. c.—the general cutaneous component of the r. oticus.

r. ot. l.—the lateralis component of the r. oticus.

r. r. u.—ramulus acusticus recessus utriculi.

r. s.—m. rectus superior.

r. sac.—ramulus acusticus sacculi.

r. sp.—ramus spinosus, or motor portion of dorsal branch of spinal nerves.

r. sp. b.—ramus spinosus of first spinal nerve *b*.

r. sp. c.—ramus spinosus of first spinal nerve *c*.

r. sp. 2. to *r. sp. 4*—ramus spinosus of second to fourth spinal nerves.

r. st. X.—ramus supratemporalis vagi.

r. v.—ramus ventralis of spinal nerves.

r. v. b.—ramus ventralis of first spinal nerve *b*.

r. v. c.—ramus ventralis of first spinal nerve *c*.

r. v. b + c.—the combined ventral rami of the first spinal nerves *b* and *c*.

r. VII. p. t.—ramus pre-trematicus facialis.

r. v. 2. to *r. v. 4.*—the ventral rami of the second to fourth spinal nerves.

rx. b.—radix brevis of ciliary ganglion.

rx. l.—radix longa of ciliary ganglion.

s. c.—subcranial canal.

sec. VIII.—secondary acoustic bundle, from tuberculum acusticum to cerebellum of same side.

sec. X.—secondary vagus bundle (Mayser), from lobus vagi to cerebellum of same side.

so. 1. to *so. 14.*—branches of the supra-orbital trunk.

sp. g.—spinal ganglion.

sp. V. t.—spinal V tract.

sp. VIII.—spinal VIII tract.

s. p. 1. to *s. p. 5.*—pores of the supra-orbital canal.

SQ.—squamosal (pterotic) bone.

sy.—the sympathetic nervous system.

sy. c.—the left commissural root of the n. splanchnicus.

sy. oph. sup.—sympathetic fibres for the r. ophthalmicus superficialis.

sy. rec. 3.—sympathetic root for the third root of the r. lateralis accessorius.

sy. 1. to *sy. 7.*—ganglia of the head part of the sympathetic chain.

sy. 1. l.—lateral lobe of the first sympathetic ganglion.

sy. 1. m.—median lobe of the first sympathetic ganglion.

t. a.—tuberculum acusticum.

t. f. 1 and *t. f. 2.*—first and second anastomosing nerves from the trigeminus to the truncus hyomandibularis.

t. hm.—truncus hyomandibularis.

t. inf.—infra-orbital trunk, containing the r. mandibularis V and the r. maxillaris and the r. buccalis, together with communis fibres.

tr. b. t.—tractus bulbo-tectalis, carrying fibres from the lobus vagi and tuberculum acusticum to the optic tectum of the opposite side.

t. so.—truncus supra-orbitalis.

t. X. 1. to *t. X. 4.*—the four trunci branchiales vagi.

u.—utriculus.

V.—the root of the trigeminus.

v. b.—ventral root of the first spinal nerve *b.*

v. c.—ventral root of the first spinal nerve *c.*

VI.—the n. abducens.

VIII—the n. acusticus.

VIII-d. l.—anastomosing root beteen the VIII and dorsal lateralis VII roots.

v. lat. VII.—ventral lateralis root of the facialis.

v. l. g. VII.—the ventral lateral line ganglion of the facialis.

v. m.—post-zonal ventral musculature.

V–VII. 1. and *V–VII. 2.*—anastomoses between the r. mandibularis V and the r. mandibularis VII.

XI.—branch of the vagus to the m. trapezius (n. accessorius).

1. l. a. e. to *4. l. a. e.*—branches of the IX nerve and of the first three branchial trunks of the vagus for the four levator arcus branchii externus muscles.

1. l. a. i.—branch of IX n. for first m. levator arcus branchii internus.

2. l. a. 1.—branch of second branchial trunk of vagus for second m. levator arcus branchii internus.

4. l. b. e.—the fourth levator arcus branchii externus muscle.

Plate I.

Figure 1.—Transection through the body of Menidia just in front of the first spinal nerve (685, cf. fig. 3), ×28. Drawn by Mr. F. W. J. Veenfliet from a Weigert preparation, after fixation in Flemming's fluid.

Figure 2.—Transection similar to the last at the origin of the hyomandibular and palatine nerves (514), ×28. The section is slightly oblique and the skin is defective on the dorsal surface.

Plate II.

Figure 3.—The cranial and first spinal nerves of Menidia gracilis, reconstructed from serial sections and projected upon the sagittal plane, × 23. The entire plot was constructed from the left side of a single specimen. No details were introduced from other specimens, though almost every point was controlled on the opposite side of this specimen and on other series of sections. The plot is drawn as if seen from the right side, the drawing having been reversed during the process of reconstruction. The correct relations would be given by the mirror-image of the plate as printed.

The outlines of the brain, eye and mouth cavity are given in black lines, the outlines of the lateral line canals in green lines. The sympathetic nervous system behind the trigeminus is omitted, also the motor component of the vagus, the general cutaneous component of the r. oticus and numerous details of the proximal portions of the cranial nerves. Compare the enlarged plot of this region, fig. 4.

All lateral line organs contained in canals are drawn as brown rings, all naked lateral line organs, "pit-line" organs, etc., as brown discs. Taste buds of the mouth cavity and lips are not drawn in. All sense organs of the outer skin supposed to belong to the communis system are drawn as red discs. The organs of the lateral lines are referred to in the text by number, counting in each line from before backwards. The scales at the top and bottom of the plate indicate the serial numbers of the sections. The latter were 15 micra in

thickness. No measurements are given in the text. These can be easily deduced from the plot.

Compare the enlarged plot on fig. 4, the isolated reconstruction of the acustico-lateral system on fig. 5 and the diagrammatic cross-sections, figs. 8 to 12.

Plate III.

Figure 4.—An enlarged projection of a portion of the same specimen as figured on the preceding plate, showing the proximal courses of the nerves, × 47. The same color scheme as in fig. 3. The transverse parallel lines across the roots of the nerves indicate the points where they leave the medulla oblongata. The ganglia are drawn with lighter shades of the same colors as used for their nerves.

Plate IV.

Figure 5. The acustico-lateral system of nerves, sense organs and canals, as seen from the left (apparently right) side, × 23. Based upon fig. 3, q. v. The outlines of the nose, eye and membranous ear are indicated and colored a neutral tint, the outline of the nasal organ being heavier in the sensory portion of the nasal sac. The canals and sense organs are colored in accordance with their innervation; thus, the r. lateralis vagi, green; the r. supra-temporalis vagi, olive green; the auditory nerve, grey; the r. mandibularis externus VII and the r. opercularis superficialis VII, yellow; the r. buccalis, red; the r. oticus, red-brown; the r. ophthalmicus superficialis VII, chocolate brown. In the canals are indicated the limits of the cranial bones to which the canals are related. All details are drawn from a single specimen, except the organs *o. 1*, *o. 2* and *man. c.*, supplied from other specimens on account of defects in the sections used for the plot.

Plate V.

Figure 6.—The lateral lines of Lophius piscatorius, 12 cm. long, natural size, seen from above. Adapted from Guitel, '91, p. 139. The several lines have been colored in accordance with their innervation, the same colors being used as in the figure of the acustico-lateral system of Menidia (fig. 5), which see. See also the text, Section 2, III. *n. a. p.*, the posterior nasal aperture. The other reference letters refer to the lateral lines; see the text.

Figure 7.—A reconstruction of the first spinal nerve, projected upon the sagittal plane, × 66. This complex includes the occipito-spinal nerves *b* and *c* of Fürbringer. The sensory component is colored yellow, the motor blue—dark blue in the case of nerves arising from the root *c*, and light blue from the root *b*.

Figure 8.—Projection of the fourth spinal nerve upon the transverse plane, × 33. This and the other diagrammatic cross-sections (figs. 9 to 12) are conventionally colored to correspond with the colors of fig. 3. They were constructed by the superposition of a series of camera lucida outlines of transections. They are diagrammatic, but not hypothetical, since no components are entered save those actually observed in the sections.

Figure 9.—Similar projection to exhibit the composition of the vagus nerve.

Figure 10.—Similar projection to exhibit the composition of the glossopharyngeal nerve.

Figure 11.—Similar projection to exhibit the composition of the facial nerve.

Figure 12.—Similar projection to exhibit the composition of the trigeminus nerve.

Figure 13.—Diagram of the relations of the eye-muscle nerves of Menidia. To be compared with fig. 12 of Allis' paper, '97.

Figure 14.—Similar diagram of the eye-muscle nerves of Amblystoma.

Plate VI.

Figure 15.—Transection of the oblongata of a young specimen of Mugil cephalus L., the striped mullet, taken at the extreme caudal end of the lobus vagi, + 50. Shows the spinal V tract entering the nucleus funiculi, the caudal part of the nucleus ambiguus and lobus vagi and the cephalic ends of the paracentral nucleus and ventral cornu.

Figure 16.—Transection of Menidia at the level of the first spinal nerve *c* (714), × 50. Shows the ventral root arising from both the ventral cornu and the fasciculus longitudinalis dorsalis, also the composition of the brachial plexus.

Figure 17.—Transection of Menidia through the lobus vagi and emerging vagus roots (640), × 50. The section includes the caudal tips of the cerebellum and tuberculum acusticum.

Figure 18.—Transection of Menidia through the tuberculum acusticum and cerebellar crest (605), × 50. The section shows the central courses of the sensory and motor IX and the apparent origins of the r. lateralis vagi and the caudal root of the VIII.

Figure 19.—Transection of Menidia at the apparent origins of the VIII and VII nerves (577), × 50. The cerebellar crest fuses with the lateral lobe of the cerebellum.

Figure 20.—Transection of Menidia at the level of the apparent

origin of the V nerve (555), × 50. The motor V nucleus lies not at the point indicated by the letters *m. n. v.*, but a few sections farther cephalad in the corresponding position.

Figure 21.—Transection of the V + VII ganglionic complex of Menidia through the geniculate ganglion (529), × 50.

Plate VII.

Figure 22.—Transection of the V + VII roots of Menidia at the level at which the ganglionic complex passes through a foramen to the outer side of the cranial wall (500), × 50.

Figure 23.—Similar transection at the origin of the infra-orbital trunk (485), × 50.

Figure 24.—Similar transection farther cephalad, showing the median and lateral lobes of the first ganglion of the sympathetic chain (481), × 50.

Figure 25.—Similar section farther cephalad, showing the origin of the radix longa of the ciliary ganglion and of the r. ophthalmicus profundus (478), × 50.

Figure 26.—A projection on the transverse plane of the course of the first facial root of the r. lateralis accessorius and the mode of its origin from the geniculate ganglion. A composite constructed by the superposition of a series of camera lucida outlines of transverse sections, × 50. The numbers represent the serial section numbers at the points where they are placed. At the point marked × the second root of the r. lateralis accessorius joins the first root.

Figure 27.—A camera sketch of a preparation of the left operculum of Menidia, × 10. See the text, Section 7, IV, 2.

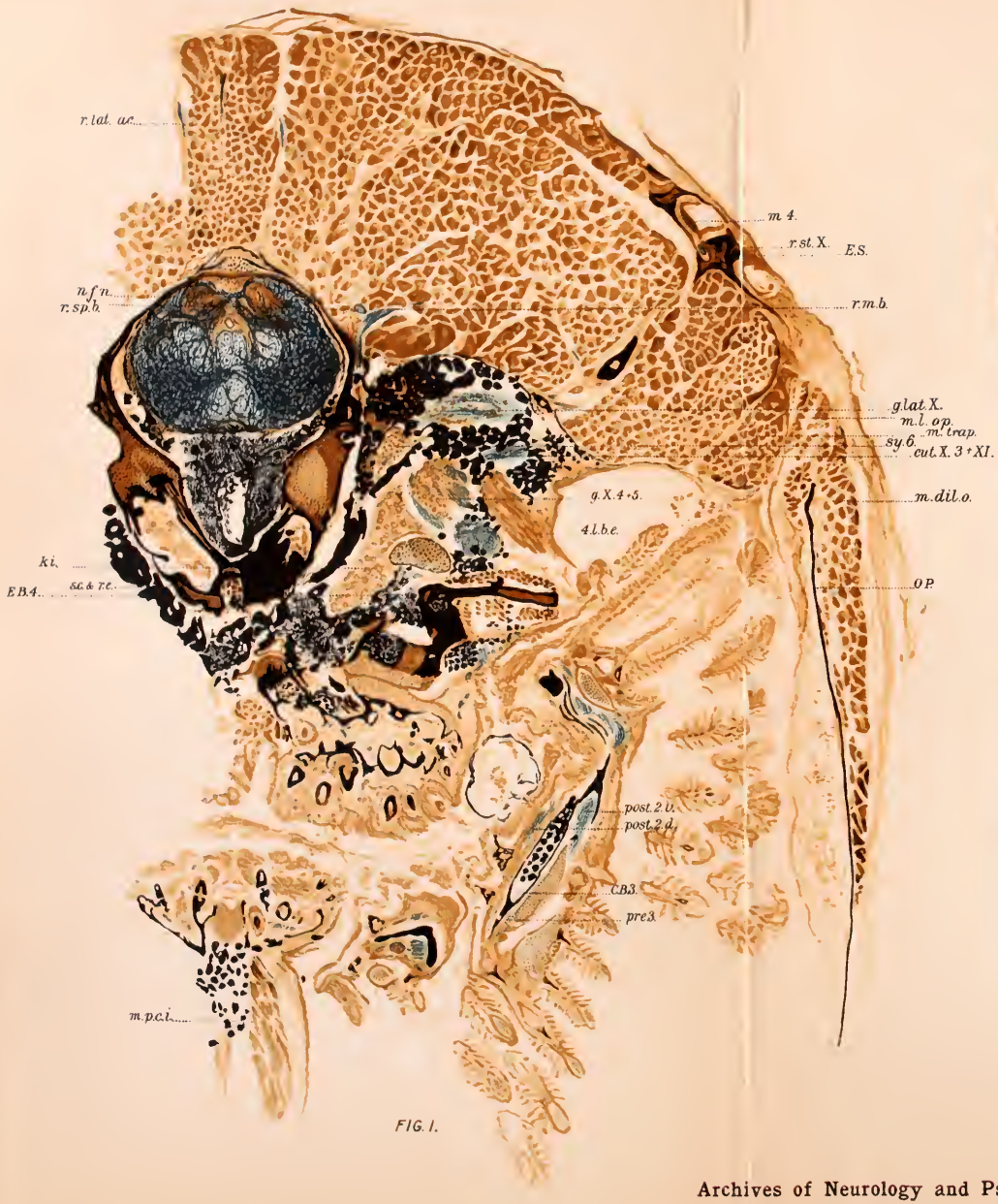

r. lat. ac.
m. 4.
r. st. X.
E.S.
n. f. n.
r. sp. b.
r. m. b.
g. lat. X.
m. l. op.
m. trap.
sy. 6.
cut. X. 3 + XI.
g. X. 4 + 5.
4 l. b. e.
m. dil. o.
ki.
E.B.4.
sc. & r.c.
O.P.
post. 2 v.
post. 2 d.
CB3.
pre 3.
m. p. c. i.

FIG. 1.

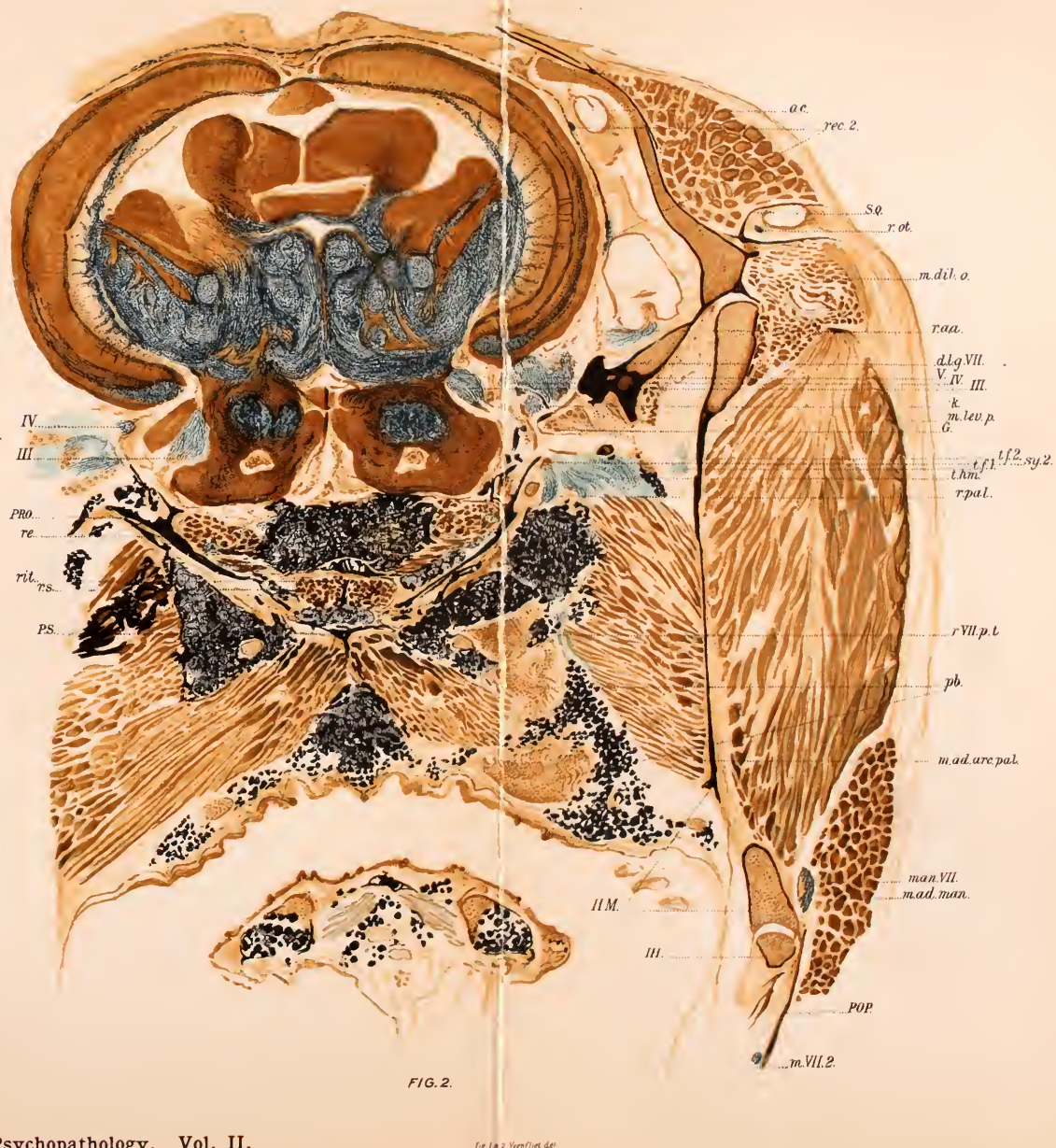
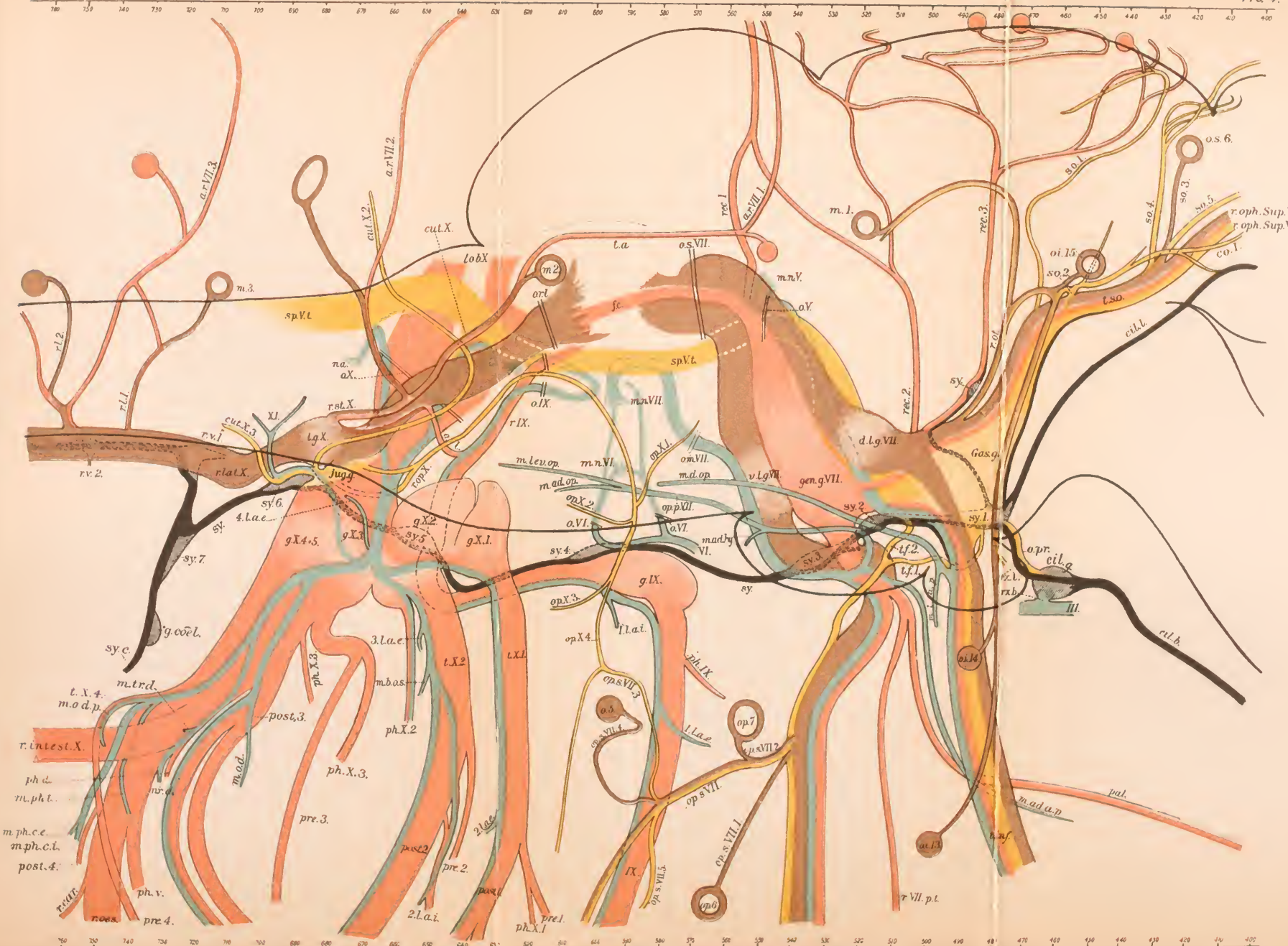
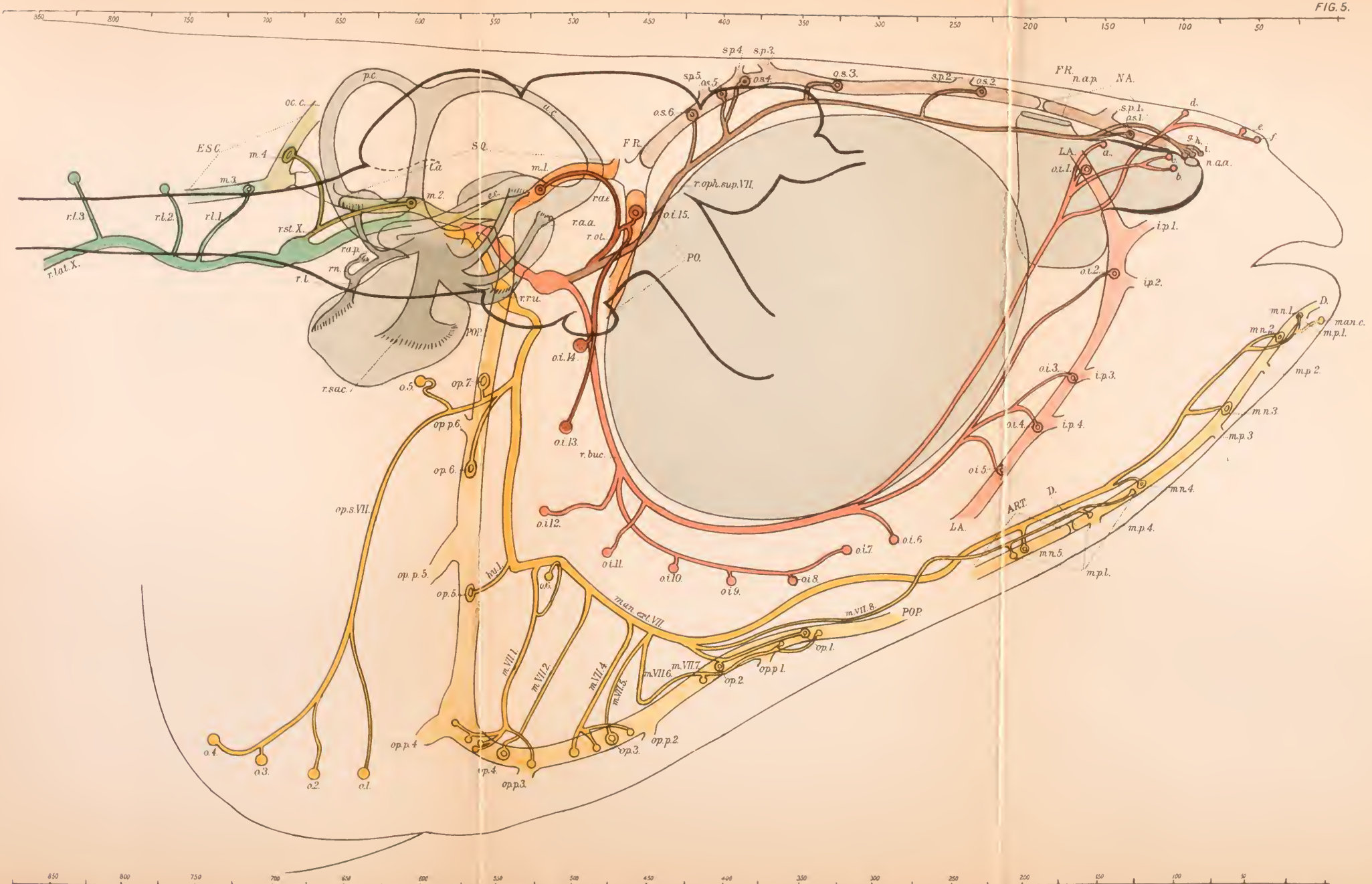

a.c.
rec. 2.
S.Q.
r. ot.
m. dil. o.
r. a. a.
d. l. g. VII.
V.
IV.
III.
k.
m. lev. p.
G.
t. f. 1.
t. f. 2.
sy. 2.
t. hm.
r. pal.
IV.
III.
PRO.
re.
rit.
r. s.
P.S.
r. VII. p. t.
pb.
m. ad. arc. pal.
man. VII.
m. ad. man.
H.M.
III.
POP.
m. VII. 2.

FIG. 2.


r.lat.ac.
a.r.VII.3.
a.r.VII.2.
ar.VII.1.
r.sp.b.
r.sp.c.
r.sp.1
r.sp.2
r.sp.3
r.sp.4
r.com.1.
r.com.2.
r.com.3.
r.com.4.
r.com.b.
r.l.1.
r.l.2.
r.l.3.
r.l.4.
d.2.
d.3.
r.st.X.
t.a.
rec.1.
rec.2.
rec.3.
gen.g.VII.
Gas.g.
d.l.g.VII.
v.l.g.VII.
r.lat.X.
r.m.1.
r.m.2.
r.m.3.
r.m.4.
r.v.2.
r.v.3.
r.v.4.
r.br.c.
cut.X.3.
cut.l.
dep.
lev.
f.d.2.
f.m.3.
f.v.3.
br.g.X.
g.IX.
rintest.X.
r.oes.
rear.
r.cerv.
v.m.
opr.VII.
m.shy
hy.1.
hy.2.
hy.3.
hy.4.
hy.5.
hy.
o.3.
o.4.
o.5.
o.6.
bc.1.
bc.2.
bc.3.
bc.4.
bc.5.
VI.
IX.
X.
cil.l.
cil.g.
cil.b.
r.s.
r.it.
r.if.
l.inf.
l.so.
r.VII.pt.
nI.
nII.
pal.
o.t.
so.1.
so.2.
so.3.
so.4.
so.5.
so.6.
so.9.
so.10.
so.12.
so.13.
so.14.
n.a.p.
n.a.a.
co.2.
a
b
c
d
e
f
g
i
h
io.2.
io.3.
io.6.
io.16.
io.18.
io.19.
io.20.
m.am.
m.VII.1.
m.VII.2.
m.VII.3.
m.VII.4.
m.VII.5.
m.VII.6.
m.VII.7.
m.VII.8.
m.VII.9.
m.VII.10.
m.VII.11.
m.VII.12.
m.hy.
m.in.
man.VII.
man.V.
V-VII.1.
VII.2.
m.am.io.13.
THE GENERAL CUTANEOUS SYSTEM.
THE COMMUNIS SYSTEM.
THE MOTOR SYSTEMS.
THE ACUSTICO-LATERAL SYSTEM.
THE SYMPATHETIC SYSTEM.
THE LATERAL LINE CANALS.

FIG. 4.


a.r.VII.3.
a.r.VII.2.
cut.X.2.
cut.X.
lobX
m.2.
orl
t.a.
o.s.VII.
a.r.VII.1.
rec.1.
m.1.
rec.3.
m.3.
spV.t.
m.n.V.
oV.
fc.
r.l.2.
r.l.1.
r.v.1.
r.v.2.
cut.X.3.
XI.
r.st.X.
n.a.
aX.
g.X.
r.lat.X.
jugg.
o.IX.
r.IX.
m.n.VII.
rec.2.
d.l.g.VII.
Gas.g.
sy.
r.ot.
so.1.
so.4.
so.3.
so.5.
o.s.6.
r.oph.Sup.VII.
r.oph.Sup.V.
co.1.
oi.15.
so.2.
t.so.
cil.l.
m.lev.op.
m.n.VI.
opX.1.
o.m.VII.
m.ad.op.
m.d.op.
v.l.gVII.
gen.gVII.
opX.2.
op.p.VII.
o.VI.
madd.y VI.
sy.2.
sy.1.
sy.6.
4.l.a.c.
sy.
gX.2.
gX.3.
sy.5.
gX.4+5.
gX.1.
sy.4.
sy.3.
tf.2.
tf.1.
o.pr.
cil.g.
sy.7.
g.IX.
opX.3.
sy.
rx.b.
III.
cil.b.
g.coel.
sy.c.
3.l.a.c.
opX.4.
f.l.a.i.
ph.IX.
t.X.2.
t.X.1.
m.t.r.d.
m.b.o.s.
ph.X.3.
t.X.4.
m.o.d.p.
post.3.
ph.X.2.
op.s.VII.3.
o.5.
op.s.VII.4.
l.l.a.2.
op.7.
op.s.VII.2.
op.14.
r.intest.X.
m.o.d.
ph.X.3.
ph.d.
m.ph.t.
inv.d.
op.s.VII.
m.ad.u.p.
pal.
m.ph.c.e.
m.ph.c.l.
post.4.
pre.3.
2.l.a.c.
pre.2.
post.2.
IX.
op.s.VII.1.
au.13.
h.mf.
r.ext.
ph.v.
r.oes.
pre.4.
2.l.a.i.
post.1.
pre.1.
ph.X.1.
op.6.
r.VII.p.t.


sp.4.
s.p.3.
sp.5.
os.5.
os.4.
os.3.
s.p.2.
os.2.
FR.
n.a.p.
NA.
s.p.1.
os.1.
os.6.
FR.
d.
e.
f.
g.
h.
i.
LA.
d.
o.t.1.
a.
b.
n.a.a.
r.oph.sup.VII.
o.i.15.
i.p.1.
ESC.
m.4.
m.3.
m.2.
m.1.
r.l.3
r.l.2.
r.l.1.
r.st.X.
r.lat.X.
r.a.p.
rn.
r.l.
r.a.l.
r.a.a.
r.ot.
PO.
o.t.2.
ip.2.
r.r.u.
POP.
o.i.14.
r.sac.
o.5.
op.7.
D.
mn.1.
max.c.
mn.2.
m.p.1.
o.i.3.
i.p.3.
m.p.2.
op.p.6.
o.i.13.
r.buc.
o.t.4.
i.p.4.
mn.3.
m.p.3
op.6.
o.i.5.
D.
mn.4.
op.s.VII.
o.i.12.
ART.
LA.
o.i.6
o.i.7
m.p.4.
mn.5.
o.i.11.
o.i.10.
o.i.9.
o.i.8.
m.p.l.
op.p.5.
op.5.
hm.l.
ob.
mun. ext.VII.
m.VII.8.
POP.
op.1.
m.VII.1.
m.VII.2.
m.VII.4.
m.VII.5.
m.VII.6.
m.VII.7.
opp.1.
op.2.
opp.2.
op.p.4.
vp.3.
vp.4.
opp.3.
o.4.
o.3.
o.2.
o.1.

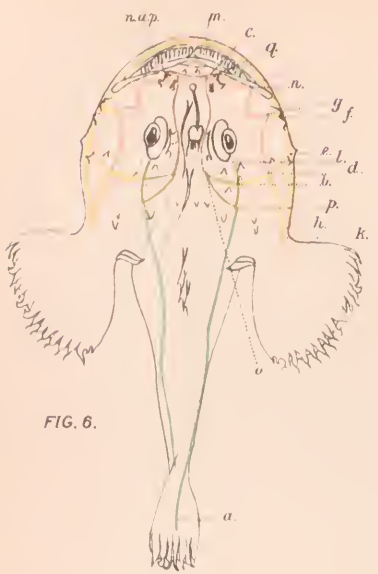


FIG. 6.

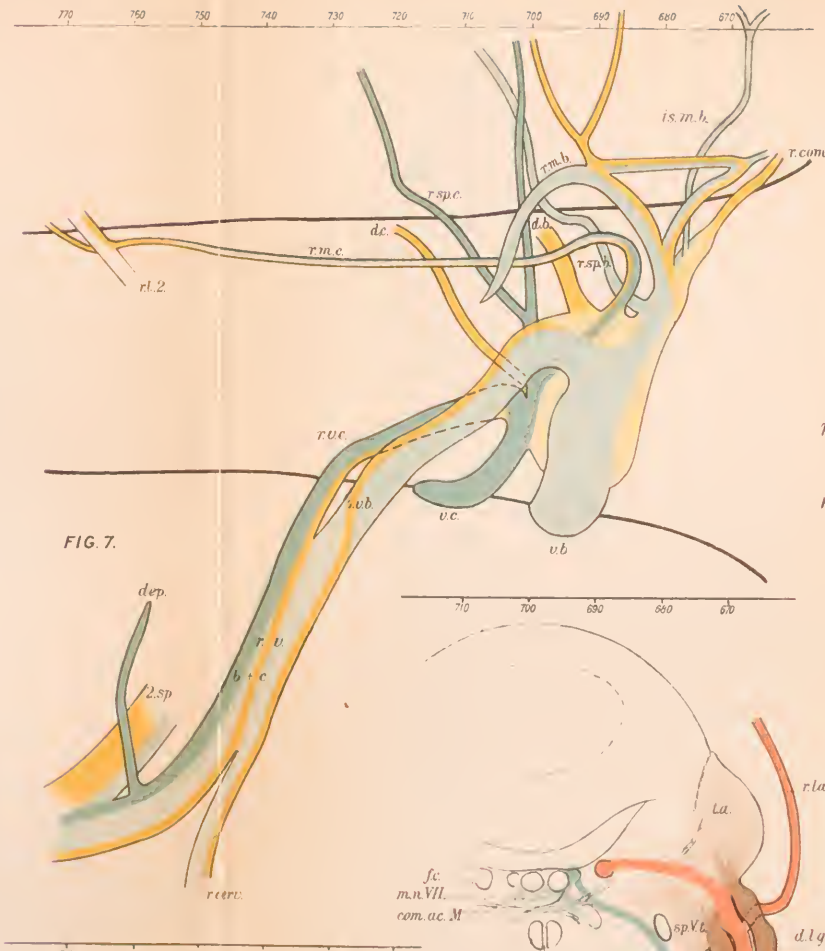


FIG. 7.

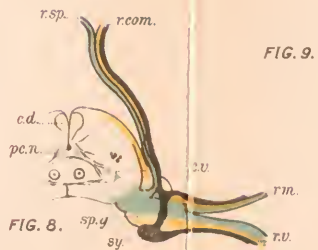


FIG. 8.

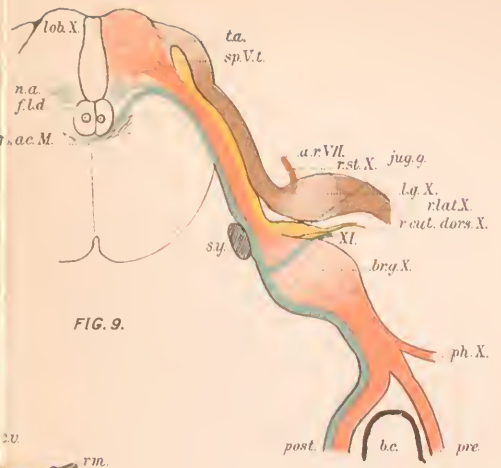


FIG. 9.

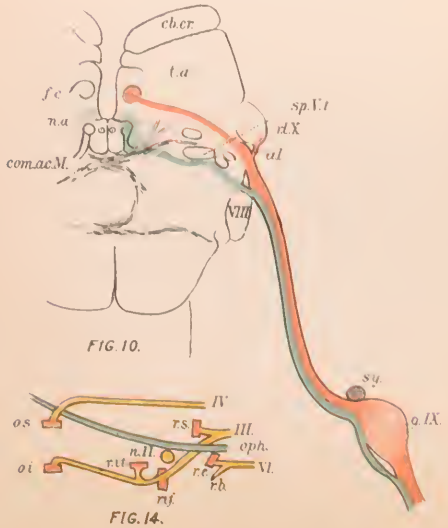


FIG. 10.

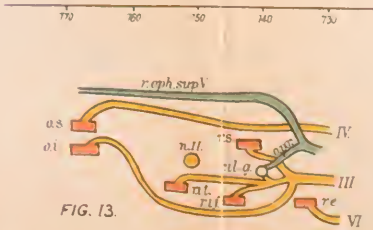


FIG. 13.

FIG. 14.

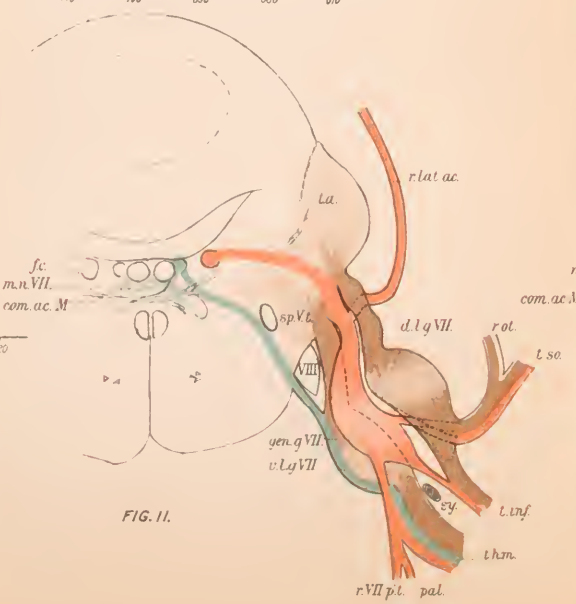


FIG. 11.

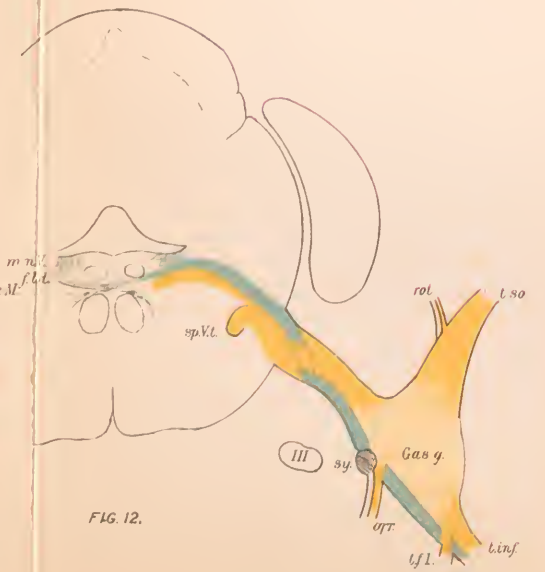


FIG. 12.

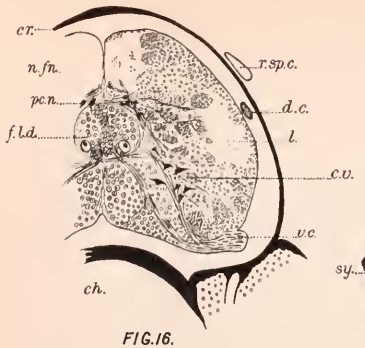


FIG. 16.

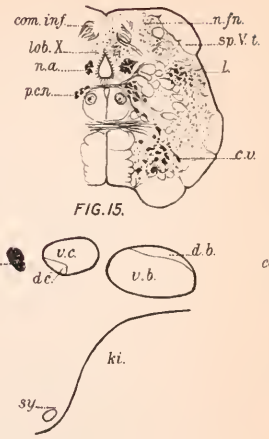


FIG. 15.

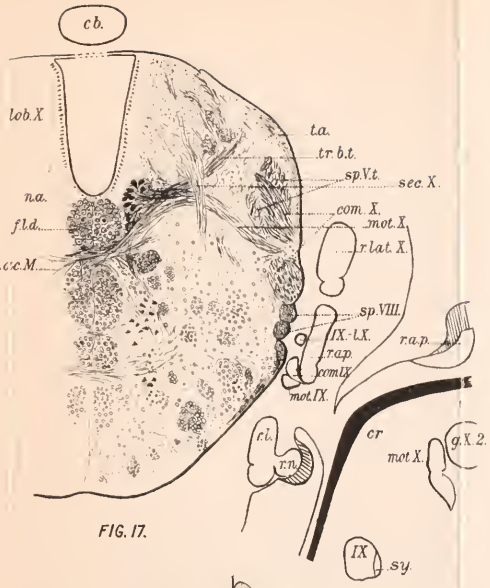


FIG. 17.

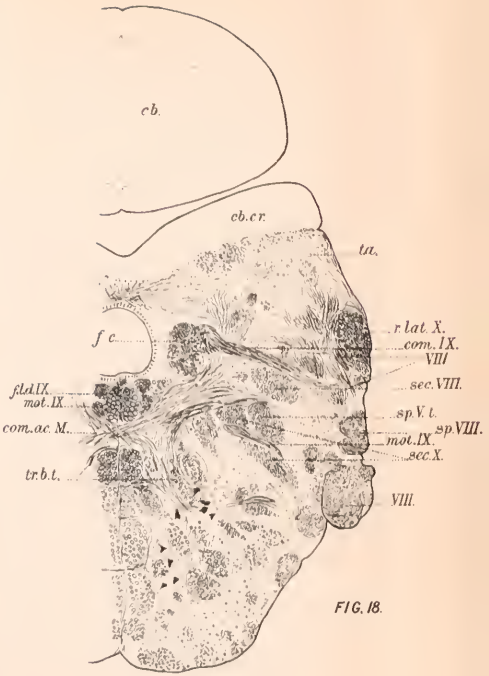


FIG. 18.

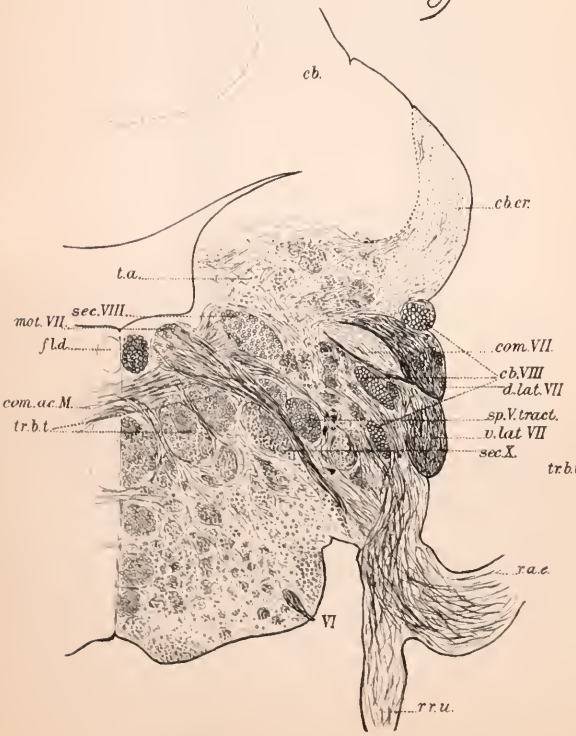


FIG. 19.

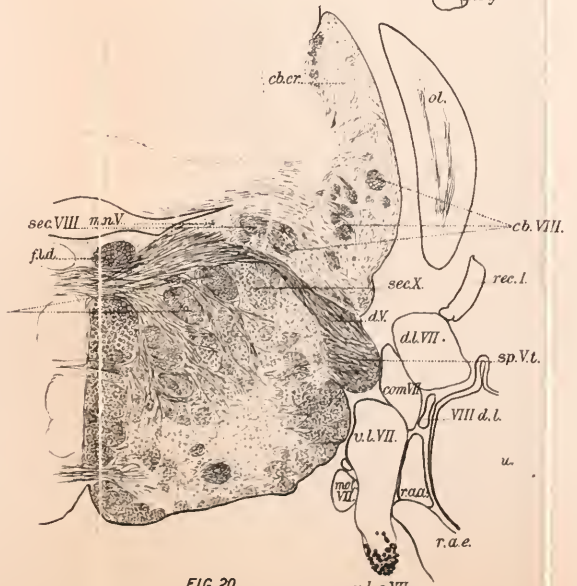


FIG. 20.

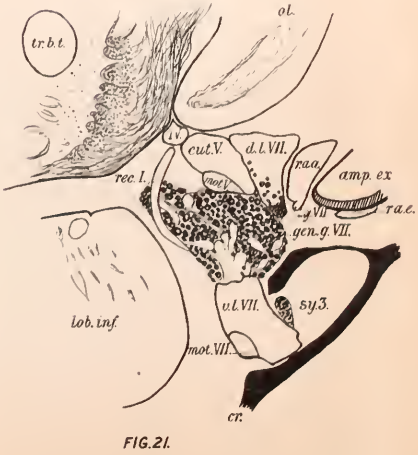


FIG. 21.

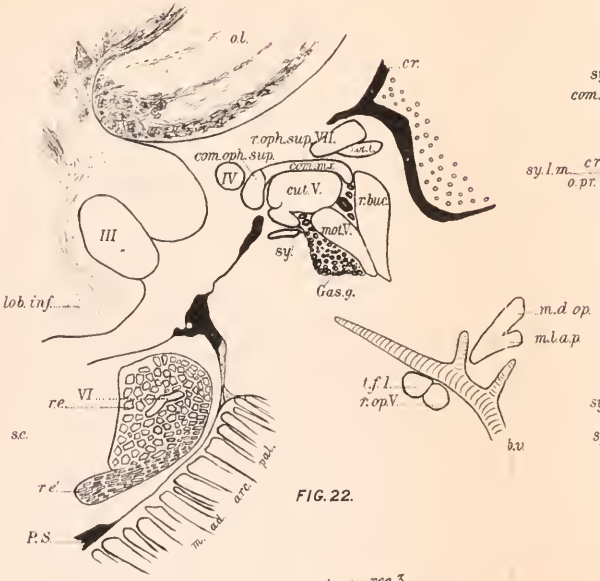


FIG. 22.

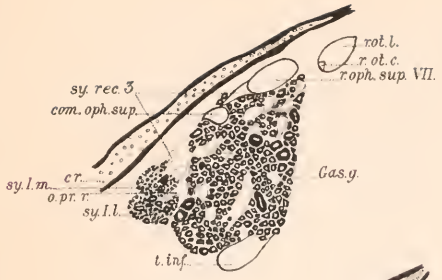


FIG. 24.

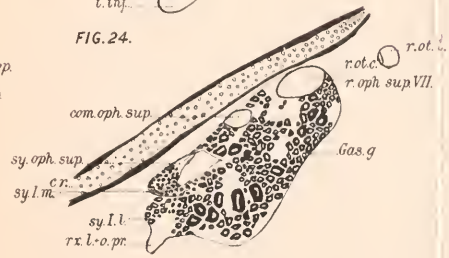


FIG. 25.

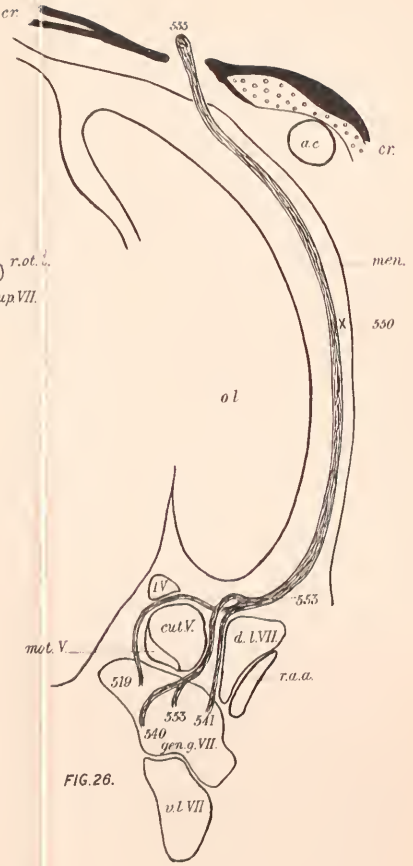


FIG. 26.

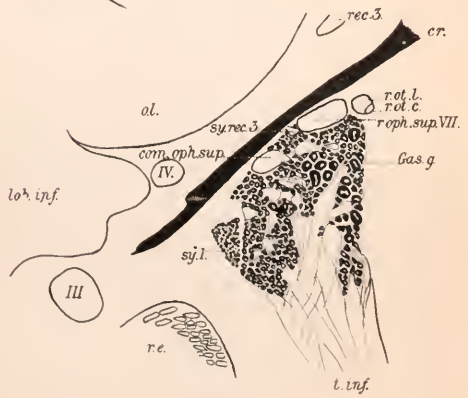


FIG. 23.

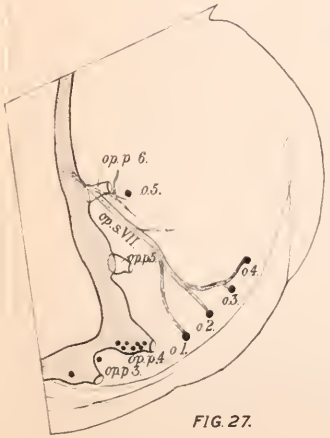


FIG. 27.

VOL. II 1899 NOS. 3-4

ARCHIVES
OF
NEUROLOGY AND
PSYCHOPATHOLOGY

616.853

CLINICAL STUDIES IN EPILEPSY.

L. PIERCE CLARK, M. D.,
First Assistant Physician, Craig Colony for Epileptics, Sonyea, N. Y.

PREFACE.

The three papers presented herein comprise the first of a series of clinical and pathological studies in epilepsy, which have been in progress during the past four years. During this period I have published in various journals several contributions on epilepsy, as soon as the contributions were finished. Although such studies were considered in the light of their correlation, nevertheless, it seems best now to abandon some of the advantages of immediate publication of isolated articles, and, by withholding from publication for a time, secure the greater benefits of surveying them by comparison, and co-ordinately and coherently presenting them in a series of studies.

Thus in the first of the series, we have dealt with Exhaustion-Paralysis in Epilepsy. First, we have given a brief historical sketch of the phenomenon, followed by a discussion of the various hypotheses and theories of

exhaustion-paralysis both in general and associated with epilepsy, together with illustrative cases from literature. We have then presented a synopsis of the recorded cases of exhaustion-paralysis in epilepsy, with a critical digest; and, finally, are recorded the investigations of a series of typical and anomalous illustrative cases from our own experience with certain conclusions from the study.

The second and third papers deal respectively with the comparatively rare association of Paramyoclonus Multiplex and Epilepsy and the still rarer clinical phenomenon, the Hypertrophic Form of Infantile Cerebral Palsy (associated with epilepsy in the author's own cases). The same general scheme of presentation has been held in mind throughout all three papers.

Assuredly, such a plan is useful to the reviewer in collecting the subjects in a series of studies, and in monograph form it is also valuable to anyone attempting to present a wide range and sequence of the clinical facts. Several of the author's cases of exhaustion-paralysis have yielded opportunity for study after death, and these investigations, nearing completion, will afford a continuation of these studies in the patho-anatomical direction. On the clinical side these studies are to be continued, with observations on other manifestations of exhausted states in epilepsy, seen in various secretory and vaso-motor phenomena, and those especially seen in the more or less complete expenditure of nervous energy of status epilepticus. Studies will also be presented on the trophic disturbances associated with epilepsy.

I.

EXHAUSTION-PARALYSIS IN EPILEPSY.

Introduction.

Post-convulsive paralysis in epilepsy is a fairly frequent phenomenon and deserves to be better known. Instances of the condition are not so rare as one might be led to believe by the absence of reference to it in works on nervous diseases, and particularly in those on epilepsy.

In a review of the literature in the text-books on nervous diseases upon the post-convulsive disorders in epilepsy, I find no mention of the condition in some, and in many others but a few random remarks upon this interesting sequela of an epileptic paroxysm.

The subject has never been thoroughly studied by American writers, although a few cases have been placed on record by them. There is not yet in any language a résumé on the subject of exhaustion-paralysis.

Strümpell[1] dismisses the condition with the statement that the affection may manifest itself after an attack, but that it speedily vanishes in all "pure epilepsy." No mention of post-convulsive paralysis is made in the chapter upon epilepsy in the American Text-book of Nervous Diseases[2]. Osler[3] states that paralysis after an epileptic fit is rare. Ormerod[4] makes no mention of the phenomenon. Neither does Hirt[5] mention the condition.

If the etiology of epilepsy is to be better understood, and if we are to draw therapeutic deductions from its case study, we must be prepared to meet the complex symptoms in detail.

Numerous observers have witnessed the paroxysms of

epileptics, but only since the discovery of cerebral localization have we made intelligent studies upon the aura, order of muscular invasion, and the sequence of the serial order of the paroxysmal phenomena of an epileptic fit.

Again, there is no longer doubt in the mind of the clinician who has given thought to the subject of epilepsy, that there are a few cases presenting unusual facility for study of the disease. It is only by carefully selecting these few cases from the great number of epileptics and by studying them closely that we make substantial progress in the knowledge of the disease. Besides, in patiently studying epilepsy in its varied phases, we give signal aid to the advancement of knowledge all over the broad field of neurology and psychiatry.

Having spent several years exclusively with epileptics, the author has not infrequently noticed cases of classic epilepsy behaving at times like traumatic, or partial or incomplete epilepsies; at such times, as it were, when the mask is off, exceptional opportunities are presented for the study of the comitial disease. This paper is largely on such cases; directed and guided by the hope that in this way we may get nearer to the true cause lying back of paroxysms of classic epilepsy.

Diverse lesions of the brain may cause clinical pictures akin to true epilepsy, and we should not force the analogies too far to explain classic epilepsy in the findings of the so-called secondary cases of epilepsy with tumors or destructive lesions for its cause. Within general limits it may be said, we should try to study and understand idiopathic epilepsy by its own phenomenon, difficult as it always is and will be.

The study of exhaustion-paralysis in epilepsy is largely a study of dynamics of the epileptic state. It is but one

of the many phases of exhaustion seen in vaso-motor, circulatory, respiratory and secretory disorders after epileptic seizures.

Chapter I.

HISTORICAL SKETCH.

As there is not as yet in any language a résumé of the subject of exhaustion-paralysis following epilepsy, it will be well to arrange the facts in chronological sequence as an aid to the development of the subject.

Aretaeus and Cassius Felix (97 A. D.) were aware that lesion of one side of the brain produced paralysis of the opposite half of the body.

Not until about the middle of the 17th century was it known (Nipper) that apoplexy of the brain was hemorrhagic.

It would appear that Audry, Thouret, and Saillaut, in the eighteenth century, had noticed the phenomenon of transitory paralysis following epilepsy. The data are not accessible either in the original or at second hand. A third observer, Pereboom, records the same fact in connection with puerperal eclampsia in his own wife. These facts were not known by Bravais.

Coming to the present century we find a case recorded as early as 1818 by Blaud, several years before Bravais' work was published. No neurologist appears to have noticed that the term *épilepsie hémiplégique* used by Bravais, has no reference to paralysis but simply denotes the unilateral character of the convulsions. Todd, on the other hand, in his term *hemiplegic epilepsy*, refers to that form of convulsion followed by paralysis. This discrepancy has produced no little confusion in the minds of subsequent writers. A number of cases of épilepsie hémiplé-

gique, so reported, surprise the investigator by containing no allusion to paralysis. But Bravais was well aware of the paralysis as an accident in unilateral epilepsy, and records at least one case (cited by Dutil).

Between Bravais and Todd we do not find a single contributor on our subject. Several epileptologists like Delasiauve absolutely ignore it. Todd was probably familiar with the subject as far back as the forties and was evidently an independent discoverer, and without doubt ought to receive all the credit for contributing our present knowledge of post-epileptic paralysis. He originated the term epileptic hemiplegia, for Bravais meant something quite different by his corresponding term. Todd reported more cases than any other writer at the time of his work and proposed the exhaustion hypothesis.

Although Todd taught his views much earlier, they were not published until during the sixties. During this decade Russell in England and Robertson in Scotland each reported several cases, and the latter professed allegiance to the ideas of Todd. In France about this time, Moreau was in charge of the Salpêtrière and according to Féré, recorded temporary paraplegia after epilepsy, but the date and reference are not given. In America, during the sixties Echeverria was aware of the fact of post-epileptic temporary paralysis and there is every reason to believe that he was an independent authority, because he had before 1870 (date of his work) kept records of 306 cases of paralysis in epilepsy, six of which were transitory. He controverts the teachings of Reynolds, that epilepsy has no connection with paralysis, but does not cite Todd as an authority.

In the seventies, the entire subject received an impetus from the new study of localization. Hughlings-Jackson

was very active during this decade and paid considerable attention to the weakness and paralysis which follow the so-called Jacksonian spasms. In France, Charcot, or rather his junior, Pitres, was equally industrious in observing and collating cases, and in making experiments which appear to prove the exhaustion theory. In Germany, Hitzig and Griesinger reported isolated cases.

At the beginning of the eighties, Jackson wrote his elaborate theoretical paper which probably gave considerable impetus to the study of the subject. Gowers gave much attention to the post-paroxysmal form in epilepsy, and took exceptions to Jackson's "relative diffusion" theory; he also introduced the idea of "inhibitory paralysis" to explain attacks where paralysis appears to replace convulsions. In France, Dutil published a monograph and Eon and Greffier wrote valuable theses on the subject. Hughes reported a case in America as a document on Jackson's teaching.

Since the early eighties, and up to the present day there has been comparatively little added to the subject. Many authorities almost or quite ignore the phenomenon, and it appears almost forgotten. Féré alludes to it a few times and recites one or two cases. At long intervals a few cases have been reported. The recent work of Binswanger on epilepsy does not do justice to the subject, and it seems a most opportune time to prepare a résumé worthy of the labor which has been devoted to the study of epilepsy by the clinicians of the past.

Chapter II.

EXHAUSTION-PARALYSIS IN GENERAL, BOTH IN EPILEPSY AND INDEPENDENT OF EPILEPSY.

We shall first attempt to collect accounts of cases of exhaustion-paralysis following conditions other than epi-

lepsy; and second, to find a basis for our theory of exhaustion-paralysis in general.

Under the first head we find, as might be expected, abundant evidence that temporary exhaustion-paralysis occurs after uræmic and puerperal convulsions, infantile eclampsia, local tetanoid spasms, the tetanoid epilepsy of Prichard, status epilepticus, hystero-epilepsy, simple hysteria, extreme acts of over-exertion (*not* examples of professional neuroses), both in connection with occupation and aside from occupation, and possibly from acute exhaustion consequent upon venery and masturbation, although a large element of doubt enters into these last two categories. *We think there is abundant evidence that acute exhaustion from any cause produces more or less severe paralysis or paresis, and that an investigation of all kinds of great and sustained effort may show that paresis at least may be temporarily present.*

When it comes to formulating a theory, however, the way is not so clear. There are many points of interest which may be enumerated. Jackson seemed to think that his cortical epilepsy was of a nature to produce exhaustion-paralysis, while the typical grand mal ran into coma instead. This idea is completely exploded by the fact that the same temporary paralyses occur in the status epilepticus along with the stuporous state.

The experiments of Franck upon cats seem to settle once for all that superexcitation of the cortex actually does produce exhaustion-paralysis, leaving the question for future study to decide what the irritants in disease states may be that cause such extraordinary liberations of nervous energy as seen, for example, in the convulsions of epilepsy.

Jaccoud, as far back as 1864, decided that *all* par-

alysis is essentially exhaustive, dependent upon overexcitation. He cites experiments which cover all the ground excepting that afterwards determined by Franck. That is, he showed that electric excitation of all nervous tissue aside from the cerebral, will produce exhaustion, respectively of vaso-motor nerves, peripheral nerves, spinal centres. Hence it would seem that experimentally there is nothing more to be done. However, Jaccoud had especially in mind reflex paraplegia from the clinical standpoint, and does not apply his theory to subjects like epileptic temporary paralysis.

Braid's view that the first stage of hypnosis was due to exhaustion-paralysis of the levator palpebræ from prolonged staring is of passing interest.

Briquet, the great authority in his day on hysteria, clearly regards exhaustion as *one* of the causes of hysterical paralysis.

We have searched the reports of occupation-paralysis but as a rule the cases seem to have no element of *acute* functional exhaustion, and are to be better explained by myopathies, neurites, or perhaps central organic disease. Nevertheless, a very few cases appear to range themselves under pure exhaustion of nervous elements.

The remarks of Féré, are of much interest, for he introduces a new element, suggestion, to explain some cases of exhaustion. He also suggests that all functional paralyses may possibly be hysterical, even though they occur in strong men. While he puts forth these views as possible, he does not appear to give them much credit.

In conclusion, it seems that some recent writers on neurology no longer have a classification of functional paralysis. This fact has puzzled us not a little. Their

classifications of cerebral, spinal and peripheral paralysis do away with the idea of functional paralysis. We have looked through a number of works on paralysis which do not index the term temporary paralysis at all. Possibly these authors, like Féré, may regard temporary as synonymous with hysterical.

With regard to the theory of inhibitory paralysis, we find the views of Gowers most prominent and they are representative of the "inhibitionists." Gowers bases his views upon the fact that when a local convulsion is aborted by constricting the limb, the resulting paralysis is more marked than otherwise, and that paralysis sometimes occurs when no convulsions have apparently supervened. Much evidence for the inhibition theory is still needed while all the paralytic phenomena are explainable upon exhaustion of nervous elements. Most writers of the day accept the latter theory for post-epileptic paralysis. Nevertheless, in passing it is interesting to note the work of Löwenfeld who is an inhibitionist for all kinds of temporary paralysis.

As the physiological *raison d'être* of exhaustion-paralysis so-called, rests largely upon Franck's experiments on the cortical area of dogs, by virtue of which it appears that overstimulation of a centre is followed by paresis, so it is well to note that other physiologists appear to obtain different results, or are, at least, able to derive material for very different theories.

Löwenfeld does not once mention Gowers' name but makes free use of the term *Hemmungslähmung* which is of course equivalent to inhibition-paralysis. He may or may not have heard of Gowers' views upon this subject, but it is certain that he has given much thought upon the matter of transitory epileptic paralysis, not only from

the standpoint of clinical observation but from experimental physiology as well.

Löwenfeld purposely confuses the subject by making no distinction between post-convulsive paralysis and those forms of paralysis which do not stand in that particular relationship with convulsive paroxysms. He labors to prove that the post-convulsive paralysis is but a single insignificant phase of a paralysis which may precede, accompany or replace a convulsion.

It requires no little effort of the imagination to bring under one classification the exhaustion phenomena which follow a Jacksonian spasm and the same conditions which result from status epilepticus. It appears that so far no epileptologist has studied this resulting loss of power from the double standpoint of a mere local spasm with retained consciousness and an exhibition of culminating serial attacks with profound stupor (and incidental loss of power in certain limbs) as demonstrated after reanimation.

Löwenfeld is so thoroughly imbued with his theory of paralysis as an equivalent of convulsions that it is necessary to read his papers very attentively in order to recognize the phenomena of exhaustion, because he never makes use of the idea of exhaustion, simply mentioning the loss of power after convulsions but not in any way differentiating it from loss of power before or during or independent of an epileptic attack. He bases his inhibitory theory on experiments by Heidenhain which other authorities on exhaustion paralysis seem to have neglected.

Without going astray too far in considering exhaustion-paralysis, it is interesting to note the work done upon paroxysmal paralysis of epileptic nature without mus-

cular convulsions preceding the paralytic state. We find H. Higier (Warsaw)[6] states that the paralytic form of Jackson's epilepsy is much less known than other epileptic equivalents. He gives a case, but the paralysis was of the intercurrent type, not appearing until six weeks after the convulsion. Since temporary intercurrent paralysis may occur with other central disturbances (syphilis, tabes, multiple sclerosis) this phase of disease is not so easily connected with epilepsy as is exhaustion-paralysis.

After discussing this intercurrent form Higier states that most attacks of typical Jackson epilepsy are followed by a short paralytic stage, which may even succeed to cases of sensory epilepsy. Sometimes we find a simultaneous occurrence of convulsive and paralytic phenomena in different muscles.

Higier quotes Löwenfeld as having gone extensively into the rationale of post-paroxysmal paralysis in his work on Jacksonian epilepsy (Beiträge zur Lehre von der Jackson' schen Epilepsie, *Arch. f. Psych.*, Bd. XXI, H I and 2) in which he states that Jackson holds that the irritation of the cortical centre which causes an accumulation of nerve energy finally discharging like a Leyden jar. He mentions that Todd and Robertson hold the expenditure of nervous force required for the convulsions is followed by exhaustion. Higier favors the inhibition theory and not the exhaustion theory because severe, even serial convulsions are not always followed by paralyses. Further, phenomena often succeed to the convulsions which savor rather of excitement than exhaustion. For example, in one of Löwenfeld's cases a hand which was left in a paretic state by a convulsion was placed about an object and clinched it for a half minute. This phenomenon had also been noted by Girard. This writer quotes

Gowers wrongly as rejecting the exhaustion theory in entirety in favor of the inhibition theory.

Again, Higier[7] describes a case of periodic paralysis not consecutive to convulsions. He refers to the experiments of Sherrington (1893) and Mislawski (1898) to the effect that if a given portion of the cerebral cortex is electrically excited some muscles act spasmodically while others are relaxed; if flexors contract, the opposing extensors are relaxed. He therefore concludes that if the intensity of the cortical irritant is of low degree paralysis may result instead of convulsion. This may in fact be demonstrated in part by experiment.

Higier as well as Löwenfeld while laboring with the best of intentions, have been led astray time and time again in denying exhaustion from convulsions a causative element in post-paroxysmal paralysis of epilepsy. They both frequently quote the well known cast-off apparent truth that the exhaustion-paralysis seen in epilepsy can not be thus caused because a strong, healthy man doing a hard day's work is not paralyzed by exhaustion, although doing much more exhausting work. They seem to be unaware that diseased and normal expenditure of force can not be compared. We regret to add that this mistake is not entirely confined to these authors in considering the disorders of motility and their consequences.

J. W. McConnell[8] discusses transient paralysis as an epileptic equivalent. McConnell says these paralyses are "sufficiently well known to require little more than mention." They may be monoplegic or hemiplegic and due either to exhaustion or inhibition. Paralysis preceding convulsion and intercurrent paralysis are much less common. The former may be due to syphilis, tumor, etc. Of the intercurrent variety a case is reported by

McConnell. These intercurrent cases of temporary paralysis are the hardest to explain, both in connection with and outside of epilepsy.

Most of McConnell's article is taken up with paralysis in the epileptic interval, and hence is not related to exhaustion-paralysis. In theorizing further McConnell concludes that this form of paralysis may be due to auto-intoxication. He further discusses family periodic paralysis which he thinks akin to these interparoxysmal palsies and likewise autotoxic by nature.

It is of considerable interest that Geo. W. Wood, an American clinician, recogized and described exhaustion-paralysis at a time when it was ignored by the profession (before Todd re-discovered it?). It is interesting to see how the passing theory of to-day is taken up to explain the facts of yesterday. The inhibitionists, Higier and Löwenfeld, had to content themselves with the loose term *inhibition*, for explanation of transient palsy in epilepsy while McConnell is able to use *autotoxics*.

Not desiring to suppress any chance of explaining the transient paralyses of epilepsies Löwenfeld's opinions will be dilated upon at greater length in another chapter.

Chapter III.

CASES FROM THE LITERATURE ILLUSTRATING EXHAUSTION-PARALYSIS INDEPENDENT OF EPILEPSY.

To illustrate that exhaustion-paralysis may and indeed does frequently occur independent of epilepsy, we will look at the following cases, collected at random from general literature.

T. M. Shaw[9] reports a case of epileptiform fits, hemiplegia, recovery; which is as follows:

Mrs. N. aged 24; fits during last pregnancy. On third day after delivery a severe convulsive attack. After that

had numerous seizures usually just after menses. Neuropathic inheritance.

November 10, serial attacks; lasted several days, about one hundred fits daily. As a result left side paralyzed. The convulsions were followed by period of stupor and delirium. Regained consciousness on November 17. Ultimate recovery; occasional seizures.

Shaw regards the case as hystero-epilepsy.

R. T. Williamson,[10] in an article upon Miscellaneous Cases of Paralysis with Etiology, describes a case of serratus paralysis in a woman after confinement, who had exerted herself violently during labor by pulling with a towel. As there was sharp pain while pulling, and as the paralysis of the serratus did not appear immediately after exertion, Williamson may be correct in assuming that the nerve supplying the serratus had been bruised, with resulting neuritis. It seems possible, however, that exhaustion-paralysis might result during the pulling incidental to labor.

Unfortunately in such cases as Shaw's and Williamson's many conditions other than exhaustion might be alleged as causes.

Handfield Jones,[11] in the chapter on Spinal Paresis, says: "It is a matter of serious consideration whether excessive fatigue may not in some instances induce or at least greatly promote the occurrence of paralysis." He cites the case of a girl in poor health who walked fourteen miles at one time; from that day she lost most of the use of her limbs; and another case of a young woman, where paresis followed long walks and bathing at the seaside. In this case, however, the paresis is not described as having supervened after a particular act of overexertion, as in the first case. Jones cites, under this head, Russell's cases of post-paroxysmal paralysis in epilepsy.

Richer,[12] says that "Hysterical paralysis sometimes follows the convulsions of hystero-epilepsy but may occur with equal readiness independently of them."

We shall have occasion to see how other writers upon the same subject attempt to bring hysterical paralyses under the ban of exhaustion phenomena. The author is also inclined to urge the exhaustion theory for both states.

Hughes-Bennett,[13] reports a case in which attacks of intermittent muscular spasms, immediately followed by complete temporary paralysis, have frequently occurred during the entire life of the patient, the health in the intervals being normal.

Bennett reports at great length a very interesting case of tonic spasms in a girl of seventeen. The first fit occurred three days after birth and she has been subject to them ever since.

The patient seemed perfectly well. The face twitches a little when she is speaking; the articulation is indistinct; intelligence subnormal (unable to read or write). All special senses, reflexes, locomotion, nutrition of muscles, normal. An attack is always preceded by a peculiar sensation; then in a few moments certain muscles are thrown in a tonic spasm, and do not then respond to the will or even to a considerable degree of outside force.

When the legs are attacked the patient naturally falls. No loss of consciousness nor confusion. The contractions may be painful. After lasting some five minutes the rigidity rapidly relaxes and the affected limb becomes flaccid, useless and incapable of any voluntary movement. After another five minutes the paralysis disappears and in ten or fifteen minutes more the limb is as well as ever. These attacks usually recur many times daily, but there are often complete intermissions for a fortnight.

Bennett describes the case with extreme minuteness. He contrasts it with similar affections and says that while

it resembles tetany somewhat it is on the whole quite different. In conclusion he says: "Hence this disorder may be looked upon as a neurosis. The intermissions, periodicity and paralysis following the spasms would seem to indicate a continual irritation of the ganglionic cells of the gray matter which causes explosions as revealed by the motor paroxysms, this excess of energy being followed by exhaustion in the shape of paresis, etc."

Frank-Smith,[14] in an article upon Hephæstic Hemiplegia (Hammer Palsy) says that hammer palsy differs from occupations like writer's cramp, because it is a strictly paralytic affection and involves the higher centres as well as the arm; symptoms like aphasia, ptosis, etc., are common. The hammersmiths and forgers make about 28,800 strokes a day. There is not only an overexertion of the muscles but of the seat of intellect, on account of the great care and judgment necessary to calculate distance and force. These men are ambitious to get ahead and often work overtime, and hence they break down at times. They are chiefly young and healthy men and free from taint of any sort.

Exhaustion-paralysis after uræmic and puerperal convulsions was found by Landois,[15] and he says that having carefully looked through the literature, he finds a great number of instances of uræmic paralyses.

Barthez and Rilliet speak of paralyses of an evanescent character which follow uræmic convulsions.

Townsend saw uræmic convulsions followed by paralysis of the entire right side of the body; recovery followed in a week.

Rego[16] saw temporary right hemiplegia after a case of eclampsia.

Dornbluth[17] saw a paraplegia follow eclampsia; it disappeared in two weeks.

Lequinie[18] saw a hemiplegia of one day's duration follow an attack of eclampsia.

Dunin[19] saw uræmic convulsions in children with scarlatina cause transitory paralyses.

These authors, as a rule, do not seem to have heard of exhaustion-paralysis; they ascribe the paralyses to toxæmia and probably are indirectly justified in doing so. Toxic conditions are undoubtedly present in uræmic and puerperal eclamptic states, but can only indirectly produce the temporary paralysis by being the immediate excitant to convulsive phenomena.

Chapter IV.

EXHAUSTION-PARALYSIS IN EPILEPSY.

Franck's[20] experiments upon cats (cited in Chapter II) proved that when we prolong the excitation of the induced current applied to a circumscribed area in the motor zone of the cortex of cats, the corresponding muscles are at first excited; after remaining contracted for a time they relax progressively. One of the characteristics of the phenomenon is limitation of the exhaustion to the excited centre.

If we suspend the excitation for a moment, the momentarily exhausted centre regains its excitability spontaneously. This temporary exhaustion is a fatigue phenomenon, reparable by simple repose. One of his conclusions applies directly to our study. He says these facts may explain a series of pathological phenomena, such as temporary paralysis after partial epilepsy.

These experiments on cats' brains are remarkably suggestive but *due allowance must always be made between physiological and pathological phenomena producing the same results.* The much overworked analogy between

Jacksonian epilepsy and true idiopathic seizures is a good example of a too direct parallelism.

Dr. Bramwell[21] in giving an account of the writings of Braid,[22] states that the latter induced hypnosis by causing his subjects to stare at a bright object held considerably higher than the head. When the eyes finally closed and could not be opened through the will of the hypnotized one, Braid explained the phenomenon by *exhaustion of the levator palpebræ*. He held the bright object at an angle which put the levator upon a great strain.

Briquet[23] in commenting upon the causes of hysterical paralyses includes in his work upon hysteria excessive fatigue and forced marches. Hence young girls when put by their parents under hard apprenticeship (such as laundry work), country girls after harvesting, domestics overworked by employers, and factory girls who have to work overtime, have all been attacked by paralysis immediately after the exhausting effort.

Jaccoud[24] has a theory of exhaustion which he describes in his article on reflex paraplegia, in which he attempts to show that *all* paralysis is dependent upon exhaustion, which in turn results from over-excitation. Excitation which is too violent, repeated or prolonged, must extinguish temporarily the excitability of the nervous elements.

There are on record very many experiments by Wedemeyer, Weber, Matteuci, DuBois-Reymond, Eckhard, Pflüger, and others in which electric currents were applied to the vasomotors, nerve trunks, cord, etc. At first slightly exciting, the persistent application of the current in time suspended the excitability with resulting paralysis for a time.

Notwithstanding Jaccoud's theory of exhaustion, he

does not once allude to the phenomenon of exhaustion by functional use.

Voisin's[25] peculiarly hypothetical idea of the theory is as follows:

"Finally the transitory paralyses of the face, tongue, limbs, transitory amblyopia, etc., are phenomena due to the manifestations of the cerebral circulation—anæmia or congestion—or to the modification of the protoplasm of the cellules themselves of the motor centres. The cells thus modified are exhausted, are no longer able to react as in the normal state; their functions are momentarily abolished until they are able to recuperate their vigor.

"This exhaustion of the cell is for a longer or shorter period. We have seen one patient in status epilepticus in whom paralysis persisted and disappeared several times in one day. The cell must have time to free itself from the debris and toxins caused by its exertion, and herein it resembles other parts of the economy."

After looking over all the literature upon the subject of *Occupation-Paralyses* it seems plain that the great majority of such cases cannot be regarded as examples of acute exhaustion-paralyses. They do not supervene immediately upon an extra effort, and in many instances there is atrophy of the muscles. There is also an element of neuritis in some cases.

Excluding all cases of myopathy, neurites, etc., it seems that occasionally pure exhaustion-paralysis may at once follow special acts of over-exertion in connection with the occupation; but on account of the history of constant use of the affected muscles before the special act of over-exertion, it seems impossible to furnish the necessary proof. Unusual exertion with the hammer, treadle of lathe, prolonged walking, etc., appear to have brought about paralysis in cases where there was no evidence of

primary myopathy or neuritis (see cases given by Féré, of piano playing; Russell, Frank-Smith, etc.)

It will be interesting to review here the views of many prominent authorities upon temporary paralysis following epileptic attacks.

Oppenheim:[26] "Seldom there occurs a motor weakness or paralysis (mono- or hemi-paresis) or a disturbance of speech under the form of aphasia or stammering. These transitory palsies are regarded as the expression of an exhaustion of the motor cortical centres (Todd, Jackson). The more they are pronounced and the longer they last, the greater is the probability that the epilepsy is symptomatic."

Féré's[27] comments are "that there is invariably a certain weakness of muscular power after convulsions, as shown by dynamometry (grip both feeble and slow). It varies with the gravity of the paroxysm. Sometimes equal on both sides, it is more commonly more powerful on one side corresponding to the unilateral character of the convulsions. Sometimes, however, we observe true paralysis of the extremities which takes the form of mono- hemi- or paraplegia. This paralysis is apt to affect by preference the muscles in which the spasm predominated. These paralyses are doubtless very evanescent and are less frequent in ordinary than after partial epilepsy. In some cases it is greatly limited, for example, to half of the face or tongue, or to a temporary strabismus." "After attacks of epilepsy.....one sometimes sees localized paralyses even where there has been no localization or spasm, either hemiplegia (Todd) or paraplegia (Moreau), or monoplegia."

He likens exhaustion-paralysis to the inability to move after waking suddenly from a vivid dream.

As for paralyses produced not consecutively but before or simultaneously with sensorial troubles which occur without paroxysms, Féré thinks they are better explained by localized arterial spasm than by the theory of

inhibition. The angio-spasm theory has long since been discarded by English writers upon nervous diseases.

Remarking upon exhaustion-paralysis in general, Féré says:

"Briquet in his Traite de l'hystérie has noted that hysterical convulsions give rise to loss of muscular power, and that pre-existing palsies are often increased."

Féré gives two histories of exhaustion-paralyses from exaggerated exercise and notes that strong emotions, excessive fatigue, forced marches, etc., are the causes of many so-called hysterical palsies.

He maintains that there is an important distinction between structural and functional hemiplegia; in the former the healthy side sympathizes with the affected side, while in the latter there is a compensatory increase of strength in the healthy side. The author has never found this statement of Féré to be true in his clinical experience.

After discussing the subject from all standpoints, Féré concludes that all functional paralyses are essentially exhaustion-paralyses, and that shock, suggestion, dominant ideas, etc., may be the equivalents of over-exertion in producing these paralyses.

Echeverria[28] in his work upon epilepsy propounds the question whether epilepsy has any immediate influence on coincident paralysis or whether this is an accidental symptom? He quotes Reynolds as holding the latter view, but the inferences from the cases under consideration, in which paralytic symptoms occurred subsequently to and in direct relation with the epileptic paroxysms, lead him to quite a contrary opinion.

Fournier[29] makes two forms, circumscribed or monoplegic and lateralized or hemiplegic epilepsy. Although cited by Dutil as an authority, he has hardly anything to

say of paresis or weakness. Like some other authors he uses the term hemiplegic epilepsy not to denote epilepsy followed by hemiplegia, but simply as meaning unilateral epilepsy. This use leads to considerable confusion.

Ross[30] states that "epileptiform seizures were first described by Bravais, then more fully by Jackson as partial convulsions, limited to half the body, or fits accompanied or followed by paresis of convulsed limbs caused by coarse lesions of the cortex, most frequently a gumma. The convulsions which follow spastic hemiplegia of childhood (due to unilateral atrophy of the brain) are first partial, then general."

We can clearly see that Ross has missed the essential principle of temporary paralysis in epilepsy when he states that a gross lesion or gumma lies back of the paralytic state upon which the paralysis depends. This error is shared by many other writers. In the beginning it is not so. As Jackson has so repeatedly shown, even when a gross lesion lies back of a paralytic state, the deepening of that paralysis by exhaustion in epileptic convulsion is sufficient proof that the symptoms are due to fatigue changes.

The historical notes of Bravais'[31] work on epileptic paralysis which several French authors quote, are not to be found in the thesis by that author.

He proposes the name "hemiplegic epilepsy" for unilateral convulsions because, as he says, hemiplegia means "to strike the half," not necessarily with paralysis but with convulsions or any other symptoms. He quotes Portal as saying that one-half of the body may be the seat of tonic, the other half of clonic convulsions. Bravais says that tonic convulsions do not come (at least commonly) from the nervous centres; hence they are not a part of epilepsy—not common therein.

He first speaks of paralysis as follows:

"Another index of hemiplegic epilepsy is that the affected side is paralyzed during the interparoxysmal intervals. This announces itself in various ways. Soon after a violent or repeated attack patient lies down with head toward the affected side. The entire body is slightly flexed by the action of the muscles which have not participated in the convulsion. This always takes place, either during the coma or after coming out of it."

According to Bravais paralysis is very variable in duration—a few moments, hours, days—perhaps even permanent. If the latter form set in, it may be total or incomplete. Bravais regards permanent paralysis after convulsions as merely the most extreme degree of paralysis which is usually temporary (excepting, of course, actual apoplexy). He cites in all five cases in his thesis. Of these one died during status epilepticus without having had opportunity of developing paralysis, as far as the notes state. Of the other four, all had paralysis, but in one case this was permanent. Hence there are three cases of temporary palsy, one of which was cited by Dutil. The other two cases are not cited anywhere; while the "permanent" case is regarded by Bravais as being due to ordinary causes, not to a lesion.

He makes the statement that if one arm alone or one side alone should experience temporary paralysis, one should be led to suspect epilepsy. Bravais concludes with statistics. He mentions twenty-five cases of hemiplegic epilepsy and thinks that many so-called general cases are really hemiplegic and are rare, while the latter are common. He does not appear to doubt for a moment that temporary paralysis is a natural result of all "hemiplegic" cases and that it is proportionate to the violence or frequency of the spasms. But clinical experience has since taught us

otherwise as we shall have occasion to observe in cases to follow in this study.

It is interesting to note that Sachs and Freud of our day seem not to have been aware of Bravais' remarks that many cases of so-called general idiopathic epilepsies are really hemiplegias with epilepsy as a symptom.

We see that, although Bravais first called attention to the exhaustion condition in epilepsy, he really reported but one or two cases carefully, but to him belongs the first discovery. Todd's careful description of the paralytic state made it known for many years as "Todd's hemiplegia."

Todd's[32] remarks upon post-convulsive paralysis in epilepsy "that hemiplegia which follows and is caused by epileptic paroxysms is very common." Todd proposes the name of epileptic hemiplegia (he was probably unaware that Bravais had described the same condition under the same name many years before).

He describes a typical case as follows—one of the more simple sort: "A patient has an epileptic fit and comes out of it paralyzed in one-half his body. Usually the side paralyzed has been the most convulsed, but the paralysis may occur when both sides are convulsed alike. This paralysis may last from a few minutes to a few hours, and so on up to three or four days or even much longer, and then disappear until another fit."

Todd separates cases in which epileptic seizures follow apoplexy, and also thinks that much so-called apoplexy is only epilepsy. After narrating some ten cases, he puts these questions:

"What is the cause of this paralysis? It cannot be congestion. This would be no explanation. What causes the supposed congestion?

"The only possible explanation is some disturbance of

the nervous energy, such as may be artificially produced by prussic acid. This undue excitement must be followed by an exhaustion of the nervous elements. This may occur either in the cortex or region of the corpora quadrigemina. If the cortex is affected the fits are mild, atypical. If from the mesencephalon, convulsions are very prominent. Both sides of the brain are usually implicated, but one side may preponderate. The preponderating side will be weaker and the paralysis which results will be more marked. The nutrition of the brain suffers. If the process is seated in the cortex, the mental powers and perception may suffer. If in the deeper parts, arterial hemiplegia may finally result."

Todd then describes two more cases (XI, XII) with autopsy, and says that they were not typically epileptic. The effusion present was secondary to atrophy.

According to Todd, epileptic seizures damage the brain, as especially shown in the paralytic cases. In the more aggravated forms, softening and atrophy follow. Some of these cases go on to true apoplexy. In this exhaustion-paralysis there is no element of compression, nor even of congestion. Even in status epilepticus with prolonged coma there is no evidence of compression.

Stokes, in his work on Diseases of the Heart, 1853, reports a case of pseudo-apoplexy occurring in the practice of Dr. Fleming and which was probably epilepsy. The left ventricle was much dilated.

Todd believes that nocturnal epilepsy in the elderly is often due to heart disease.

In his later teachings, Todd seemed to retract the statement that both sides of the body were equally involved in those cases with sequent paralysis of one side, and urged, as the latest teachings do, that the side most paralyzed is most convulsed even when apparently both sides participate in the convulsion.

I have seen several cases of so-called apoplexy, in subjects quite advanced in years, which were sooner or later followed by apparently true epilepsy; to all appearances such cases were typical of epilepsy with epigastric aura. They would seem to lend much evidence to Todd's statements that apoplexy is frequently mistaken for epilepsy. Doubtless much might be said on both sides of the question depending in no small way upon the viewpoint of the observer. Todd's statement about epilepsy ending in apoplexy are almost without a single proof. As good an authority as Gowers states that this sequela is "an exceedingly rare event."

Robertson[33] writes as follows: "But I am inclined to think that the late Mr. Todd was correct in supposing that severe and protracted convulsions may themselves in some instances be causative of palsy of a few hours' or a few days' duration through simply the exhausting influence exerted on the cells of the central ganglia without much if any appreciable change of tissue. This explanation is especially applicable to some of the cases of hemiplegia following epilepsy in which the paralysis passes away in a few days; in other cases, however, the symptom is more probably the result of a small effusion of blood."

We shall see that Robertson falls into the same error as Todd about epilepsy ending in apoplexy and states that the condition of epilepsy may give rise to a small effusion of blood.

Gowers[34] concludes that epileptic "hemiplegia of Todd" is most distinct after unilateral convulsions, thus confirming Todd's observation. He also thinks with Jackson that general prostration after bilateral spasm is probably analogous; severe seizures are due to exhaustion, but slight ones or none at all, in which paralysis obtains, must be ascribed to inhibition. Sensory discharges

(sensory epileptic attacks) may leave the arm powerless (motor paralysis). Loss of speech after right-sided seizures probably has an inhibition origin. Gowers makes no effort to invoke exhaustion of nervous elements without convulsive phenomena, as has been urged so frequently in paralysis following mental shock, nor does he refer to the extreme physical and local weakness in epilepsy of a psychic character.

Conclusions from Gowers'[35] monograph on epilepsy: A fit of epileptic type—very rarely general—commonly in one-half the body or affecting only a limb. The form and nature of the paralysis following such fits differ much and essentially. The most common form is a transient weakness incomplete in degree, passing away in a few hours, usually recurring after other attacks and therefore probably functional. In another form paralysis succeeds the convulsions and may persist for weeks and months, irrespective of subsequent convulsions. In this class both the paralysis and convulsions come from the same organic brain disease (such as apoplexy). There is a third class where persistent paralysis results from a hemorrhage which takes place during a convulsion. This class is *extremely rare*.

The transitory paralysis is such that the patient cannot stand or walk for some minutes. It rarely lasts long and is commonly regarded as due to general exhaustion perhaps, but not necessarily from the severity of the convulsions. Motor weakness is greater after partial or unilateral convulsions than when the latter are general. It is usually most marked in limbs where convulsions began. Todd describes it under the title of epileptic hemiplegia. The duration of the weakness is a few hours, rarely one or two days. Slight power of movement may remain,

especially in the leg. In the arm the paralysis may be absolute, motion returning in a few hours. It may follow every fit. If it is a case of recurring convulsions every few hours, paralysis may last until attack is over. In the class of fits which are most frequently succeeded by weakness, viz., those which begin in the hand or foot, paralysis often follows an extremely mild attack, and even one in which convulsions are absent.

A severe epileptic attack leaves the muscles atonic and flabby, the knee-jerk being occasionally lost, to reappear in half a minute. More frequently the reflexes are increased so that foot clonus may be obtained (Jackson). According to Beevor we may get foot clonus after every severe fit. After slight fits the reflexes are unchanged; after attacks of greater severity we may get foot clonus and increased knee-jerk. After very severe fits there may be for a very short time loss of knee-jerk followed by excess. There is no relation between the excess and severity of fit (Beevor). In the case of bilateral convulsions of apparently equal severity (but where the head deviation shows that convulsion is more severe on one side) clonus may be obtained on the side toward which the head is bent, but not on the other. Plantar reflex is abolished for from five to ten minutes after severe fits.

Occasionally there is partial loss of speech, especially after right-sided seizures, often associated with right-sided weakness (as in permanent aphasia with right hemiplegia). This loss of speech may occur without weakness, especially after slight fits beginning in the face.

There is assumed to be a discharge from cerebral motor centres. Todd believed that epileptic hemiplegia was due to exhaustion of part of the brain from excessive action, and this view has been accepted by Alex. Robertson and

Hughlings-Jackson. Jackson says that the weakness and foot clonus may be due to exhaustion of fibres of the internal capsule of the brain or of the lateral columns of the cord by the discharge through them. This may explain part of the phenomenon, but not the extreme weakness after a slight local fit. One patient after a severe general fit may have no weakness, while another with a mere local fit may be unable to move hand or foot for an hour. Jackson attempts to explain this by "relative diffusion," but this is a pure hypothesis and is unsupported by any evidences.

After giving several histories Gowers says that in cases of weakness or transient paralysis without motor spasm, there is lowered activity without discharge. These paralyses are probably inhibitory rather than exhaustive. If a commencing fit is arrested by ligature the paralysis is greater than if it ran its usual course. The ligature acts on the sensory cortex and increases its resistance or inhibitory power on the motor centre related to it.

Inhibition and exhaustion may be conjoined in a fit. The phenomena of some attacks show that while conditions of discharge and inhibition are antagonistic and in high degrees incompatible, yet they may co-exist to some extent, *i. e.* the centre may be in a state of partial inhibition and partial discharge and ultimately one state must preponderate. In no other way can certain clinical phenomena be explained.

In temporary paralysis the state of the limbs is precisely like the permanent condition in cerebral paralysis. There is motor weakness, lowered cutaneous reflex and excess of tendon reflex, etc. In repeated unilateral convulsions we may get actual hemiplegia, alike in every way to that due to organic disease.

In careful examinations of the reflexes in epileptics, during the past four years, covering more than a hundred epileptics in several hundred distinct seizures, I found the following rule to be invariable: Immediately after all fits the reflexes are abolished, remaining absent a few seconds or minutes, in all cases depending upon the amount of exhaustion of nervous elements, other things being equal. When they begin to return, that side most particularly in convulsion would be found to be in reflex exaggeration and remain so until exhaustion phenomena entirely disappeared. Reflex exaggeration is a fair index to the severity of the fit, as expressed in exhaustion phenomena. True foot clonus is rarely obtainable in epilepsy, excepting in cerebral palsy followed by epileptiform convulsions.

Jackson[36] sets forth the following theory: The rationale of the Bravais localized convulsions, occasionally resulting in palsy is as follows: The temporary post-epileptiform paralysis depends on temporary cerebral exhaustion consequent upon the excessive discharge during paroxysms. During the paroxysms there is liberation of energy from the highly unstable cells of the cortex. Currents must flow along the lines of least resistance. The spasm is a sort of resultant of numerous efferent impulses. Local paralysis occurs in parts earliest and most strongly convulsed and corresponds to an exhausted condition of the brain just as the convulsion is an expression of the discharge in the brain. As the state is merely exhaustive, we have recovery.

Some physicians believe that the paralysis is due to congestion—to an asphyxia as it were, due to interference by the convulsion with the respiratory movements; this, however, does not explain localized paralysis. Nor could

this paralysis result from small extravasation of blood, for we could not thereby explain repeated attacks in the same locality. It is, however, true that a small clot may produce local paralysis. In severe general epilepsy we do not get local paralysis, although here we have extreme asphyxia from respiratory spasm.

Some claim that paralysis is not always of the first and most convulsed parts. Jackson has always found it so. In some cases of cerebral hemorrhage the clot causes convulsions; but this is another matter.

A tumor or gumma of the cortex may, during its growth, give rise to partial convulsions followed by paralysis.

We see at times temporary paralysis not preceded by convulsions. These do not belong here.

The post-epileptic paralysis may be very slight and momentary. For example, patient may be unable to execute more complicated movements.

In discussing paralysis after general seizures, Jackson says that "such a condition may be denied, but that the helpless state which accompanies coma is not necessarily due to the latter and is to be regarded as of the nature of total paralysis. Coma is a negative psychical condition, and the prostrated physical state is a negative physical condition."

Post-epileptic paralysis is probably never complete and varies much. Some power of motion remains. After consciousness is regained there is still weakness and prostration. This is also true of the petit mal.

It might be said of local paralysis that there is a disproportion between severity of seizures and resulting paralysis. But diffuse weakness or general prostration might be equivalent to local palsy.

In estimating a discharge it will be found that the more deliberately it sets in and the more slowly it spreads, the more local it is and the longer it lasts,—so the resulting paralysis will be observed to be correspondingly more local, more complete and of longer duration.

If this is admitted, then the more deliberately the discharge begins and the slower it is, then the less diffuse are the currents and the longer the duration. Conversely the more sudden the discharge, the faster is the spread and the greater the range and the paralysis is more widespread, less in degree and more transient. If this is admitted, then the more sudden the discharge begins and the more rapid it is, the more widespread are the currents and the less they continue, and the post-epileptic exhaustion is more widespread, less extreme and more transient. The paralysis is apparently less because it is not localized; because it is slighter in degree and soon passes off,—in fact it is looked on as weakness and not paralysis.

Let us suppose two cases with cortical lesions of the same area—in one the discharge requires ten and the other two minutes. In the first case the currents might flow wholly into the area while in the second they would flow less exclusively to the arm and also to parts more generally represented, and finally to lower and collateral centres. In rapid discharge diffusion is easy, while in the slow form the same resistance is not overcome and the currents take the path of less resistance. The aggregate amount of paralysis is much more after general than local seizures.

This is essentially an epitome of Jackson's teachings, but all of his theories are so very hypothetical that anyone not particularly skilled in dealing with such matters had best not cross swords with him. This much can

be said, however, in our present-day knowledge of psychopathology that Jackson's teachings are proving themselves true.

Dutil[37] says that paralysis is rare in general epilepsy. Delasiauve and Herpin do not give a single example. Russell-Reynolds speaks as if paralysis in epilepsy was the merest coincidence, or was at best indirect in its relationship. Dutil says that in partial epilepsy it is otherwise. Convulsions of cortical origin are often associated with permanent paralysis which may or may not precede the convulsions. Sometimes the paralysis becomes worse progressively with each attack of convulsions.

There are also transitory paralyses which follow epileptic attacks. Bravais first noticed this fact, but his findings were ignored. Todd apparently made the discovery anew, but the matter was again ignored until the study of cerebral localization.

Partial epilepsy is often followed by transitory paralysis with either flaccidity or rigidity of muscles. Seat of paralysis is usually in muscles which have been convulsed. Paralysis may last from a few moments to a few weeks. This paralysis must be carefully distinguished from that which is due to lesion of the brain.

Todd's theory of partial epilepsy has been verified by Franck and Pitres[38] on animals. If motor areas are faradized, as in the dog, partial epilepsy results, and this hyperexcitation is followed by a loss of excitability which is local and transitory.

Under the title of Jacksonian Epilepsy and its Clinical Equivalents, Löwenfeld[39] states on page 5 that in those convulsions which are followed only by transitory paralysis or by no paralysis at all, the antecedent intra-cranial condition consists most frequently of cerebral tumors,

injuries to the brain and cerebral syphilis; much more rarely non-traumatic abscesses, softening and hemorrhage. Other conditions causing Jacksonian epilepsy are parenchymatous encephalitis, dementia paralytica and multiple sclerosis.

This author cites his cases and comments upon them as follows:

"Female, 39, widow, in general health until six years ago. Convulsions set in usually left-sided, beginning in hand or foot and ascending to head. Aphasia followed. No mention of actual acute exhaustion-paralysis but stated the limbs became gradually weaker. Diagnosis of multiple sclerosis."

Löwenfeld also discusses uræmic convulsions, reflex epilepsy, hysteria and finally cases of unknown origin.

"Case III, (p. 14), patient, male, 38, has a local Jacksonian spasm, often limited to a single hand; very rarely there are extensor spasms of arm and leg. The attacks last three minutes on average, and for several minutes afterward there is a feeling of great exhaustion of the whole body with tremor of the legs."

Löwenfeld says that this case is a plain one of petit mal taking the form of Jacksonian epilepsy. This patient had been a victim of sexual excesses.

"Case IV, (p. 17), a man of 49. Spasm of fingers and forearm in the region of the median nerve. After one one-half minutes the upper arm and shoulder are involved. Then the spasm returns to the fingers which are partly flexed and partly extended (thumb and index). This stage lasts twenty minutes and after it is over the patient can move his fingers only with great difficulty. While under observation a second attack followed after an interval of three days, and when it was over patient was unable to flex fingers for one-quarter to one-half an hour, and this phenomenon was observed no less than ten

successive times. In the interim there was no abnormality except a weakness of the right arm. This affection was limited to the right arm, of which a number of the muscles were in a state of paresis." The progress of this case was as follows: "The convulsion abated but the muscular paresis became worse. Eventually hemiplegia set in. Autopsy showed a tumor in the left cerebral cortex near the arm centre."

"Case V (p. 20).—A man 48 years old. Convulsive attacks had begun several years before. They appeared to be examples of Jackson spasm with other attacks like genuine epilepsy (unconsciousness, etc.) It does not appear that acute exhaustion-paralysis occurred except fugaciously, but after a year or more a constant and progressive weakness of the left arm appeared, soon followed by a similar condition in the left leg. Soon the picture of severe left hemiplegia was present. As in the preceding case there was a recurrence of the convulsive attacks as the paralysis appeared. Patient died with symptoms of intracranial pressure and at the autopsy a number of hemorrhagic gliomata were found."

"In Case VI (p. 28) the spasms were for a long time limited to the face, left side, they finally became general. There were spontaneous paresis of the left arm and signs of dementia. Patient died of status epilepticus. She was aged 34 when first seen, and her spasms had begun a year earlier. Autopsy not made."

Löwenfeld had diagnosed a cortical lesion which gradually spread at its periphery.

"Case VII.—A boy, aged 7 or 8 years. First convulsion after a prolonged bicycle ride, limited to right arm, lasted five minutes, consciousness retained. Attacks repeated, involving also the leg and head. From the time of the first attack there remained a weakness of the right arm which lasted fourteen days. These attacks were persistent and frequent, as many as twelve in a day. This weakness of the right arm which appeared to have

resulted from the first attack also served later as an aura to denote the approach of a fresh convulsion."

When examined by Löwenfeld the right hand shake was weaker and the movements of the right arm slower than opposite side. Handwriting like that of writer's cramp. Although this child was perfectly healthy to all appearances Löwenfeld and others believed the case to be one of hereditary syphilitic cerebral affection.

"Case VIII.—Male, aged 35, had an isolated convulsive attack when 27, lost consciousness and had spasms of the right arm. At intervals afterward he had attacks of anæsthesia of the first three fingers with unsteadiness and weakness of the right hand. Finally he had a return of the convulsive attacks in the right hand. It was followed by extreme weakness lasting eight days, but during that period when the hand was closed around an object, a spasmodic contraction of the fingers resulted.

The anæsthetic attacks were now resumed for a time and later a third spasmodic attack developed, with two months later a severe attack of eight convulsions of the hand in one night, with nine attacks on the succeeding day. Marked weakness of the right arm followed amounting to helplessness. Power returned in about ten days or so.

Diagnosis of this case, syphilis, infection dating back about three years. Probably a gumma in the cortex."

Löwenfeld states that aside from the convulsions in Jacksonian epilepsy there are other phenomena which are of interest. In the motor region there is but one of these phenomena to be considered, viz., the paralysis which follows the convulsions. It appears from his observations that the phenomenon of paralysis may not only follow but may even precede the convulsions—in the latter case constituting the aura. There is no definite connection between the convulsion and paralysis.

In Case VI (Löwenfeld), essentially limited to the face for a long period, a facial spasm was usually preceded by weakness of the hand, the patient dropping any article which she chanced to have in the hand. This hand was never involved in the facial spasm save to a minimal and transitory extent, limited to an occasional flexing of the finger.

In the case of the boy (VII) the aura preceding the convulsion was invariably a sudden loss of power in the hand, a fact which has been previously recorded by others. Case IV had a distinct weakness in the arm after the first convulsion, with persistence; but Case V even after powerful and repeated contractions showed no disturbance of motility whatever. In Case VI the repeated and severe contractions of the facialis left no apparent added weakness in the muscles or nerve. In Case VII, on the other hand, the very first convulsion produced a long remaining weakness of the arm, and Case VIII in the course of very many spasms showed paresis on but two occasions. Hence, Löwenfeld infers that there is absolutely no relationship between the duration and intensity of a spasm and the resulting loss of power. Löwenfeld continues:

"It appears that in certain cases whenever paresis develops, the extent of the paresis is less than that of the convulsed muscles; at other times the two coincide, while there is a third class of cases in which the paralyzed territory is greater than that of the convulsed muscles. This class occurs but seldom. It is also very rare that the paralysis increases spontaneously in place of going away.

Thus far there have been but two hypotheses to account for these post-paroxysmal paralysis. One claims that a small blood extravasation has occurred in the brain or perhaps merely an intense local congestion. This view

may be at once dismissed without argument. The other view is that of Todd, Robertson and Jackson, viz., that the paralysis results from exhaustion in a given area of nervous tissue. The discharge of nervous force is followed by a stage of functional impotence. The great disproportion between convulsion and paralysis speaks against this view which is also untenable on physiological grounds as well, because the energy released by a day laborer in a day's work is far in excess of that expended in a convulsion which lasts but a moment."

Again, there are not wanting in the post-paroxysmal stage and accompanying the paresis, evidences which point to excitement. Here may be cited Löwenfeld's case VIII (spasmodic closure of hand, etc.), and the remarks of Girard (Étude sur L'épilepsie Jacksonienne, p. 27):

"It has equally been observed that whenever a paralysis or transient hemiplegia follows a convulsion, the spasmodic state returns if patient attempts to execute a voluntary movement, or when these regions are explored."

Löwenfeld continues that "the paralysis may precede or coexist with the convulsion, or may occur in an area which has not been convulsed; and when we compare all these conditions which make against the exhaustion theory, we see that the only tenable view which is able to account for all these data is the theory of inhibition.

"The post-paroxysmal defects of speech have also been explained by the exhaustion theory; but the same objections obtain here as in the case of the convulsions of arm and leg. The speech defects may precede as an aura, and a comparison teaches that the inhibition theory is the only satisfactory mode of accounting for these phenomena.

"The sensory aura which may persist through the attack and remain after it is over is also a prominent phenomenon of Jacksonian epilepsy, and presents a close parallel to the motor phenomena. Various paræsthesiæ such as numb-

ness, tingling, formication, chilliness, etc. These sensory phenomena are associated with the motor in the same subject, in such a manner that there can be no doubt that they are correlated and due to the same causes."

An interesting phenomenon presented by Case VI was sialorrhœa; as far as Löwenfeld knows this is the second recorded case, the first having been described by Landouzy and Siredey. This phenomenon was associated with spasm of the face, tongue and muscles of the jaw, and may also be produced experimentally on dogs by irritating an area of the cortex cerebri. It appears to prove that the salivary and facial nuclei are in close apposition. In this same patient the heart's action was slowed and weakened. Francois-Franck has obtained the same result experimentally.

In Case V there was another unusual phenomena, viz., a feeling of constriction about the neck or chest due, perhaps, to spasm of the pharyngeal or œsophageal muscles from cortical irritation. The same phenomenon was also observed in Löwenfeld's Cass XII.

Löwenfeld here describes (Case IX) "a woman of 34 who first became epileptic when 3½ years old; the convulsive attack was followed by permanent hemiplegia, and the convulsive attacks gradually disappeared for longer and longer periods of time. One pause was eight months long. After a long period they became frequent again and finally amounted to six or eight daily. They were of different degrees. The more severe attacks began with a twitching of the paralyzed leg. Both the sound and paralyzed sides participate. Consciousness is lost and the attack is throughout one of major epilepsy. Another type of attack has consciousness retained, mere spasms of the paralyzed side lasting a few seconds; these may be inhibited at times by the will, or by the use of English

smelling salts. There are also rudimentary attacks of mere epigastric pressure or other vague sensory phenomena."

Under paralytic equivalents or the paralytic form of Jacksonian epilepsy, Löwenfeld cites Pitres' experiments and states that he was the first to speak of the paralytic equivalent of Jackson's epilepsy. These equivalents cannot of course be referred to exhaustion direct.

Löwenfeld quotes Heidenhain as saying that both excitement and inhibition can be due to the same agency. In experiments on dogs weak currents increase the excitability while the tonic application inhibits it.

He relates a case, which appears to show that the same cortical irritation may produce either spasm or paralysis. After two short attacks of spasm which involved the right hand only, he experienced a more complicated seizure. In the hand there was motor paralysis, spasm and sensory disturbance. In the forearm and upper arm sensory disturbance. In the facial region sensory disturbance, spasm and speech disturbance. It is thus evident that exciting and inhibiting processes are at work in the same cortical area. Slight peripheral irritation is often sufficient to call forth inhibitory acts while calling forth the flexor spasm.

Löwenfeld states that Jacksonian epilepsy may rarely develop the status epilepticus with fatal termination. He cites his own case already mentioned as well as cases of N. Weiss, and of Landouzy and Siredey as examples of this sort. I have seen numerous cases of so-called Jacksonian epileptics in status. Two of these are to be found in the chapter presenting my original cases.

In a comparison between Jacksonian and idiopathic epilepsy Löwenfeld unfortunately does not mention the

post-convulsive paralysis of true epilepsy; but he has no doubt that all epilepsy whether major or petit mal or Jacksonian epilepsy, is one and the same affection.

I believe that Löwenfeld's ideas are so thoroughly combatted by the views and evidence of the other authors whom we have quoted as well as by the evidence from my own experience, that his presentation can be dismissed without more extended comment.

Chapter V.

CASES OF EXHAUSTION-PARALYSIS IN EPILEPSY CITED IN LITERATURE.

In this and the next Chapter some sixty or more cases are recorded, all of which have been ranged by their authors under the head of paralysis, presumably temporary, of epileptic origin. It is possible that at least a third of these cases do not belong here at all, as will appear later on. This then leaves some forty-one legitimate cases, and the list may possibly need further reduction, because Jackson in several cases does not expressly mention that there was paralysis or weakness, although from the compass of the article one might infer it. Deducting three cases for safety, there remain at least thirty-eight cases. Of non-typical cases there are at least eighteen.

We may then arrange the cases as follows:

Typical	20
Atypical	18
Doubtful	5
Total	43

Typical and atypical cases may be arranged into

Simple, about	30
Complicated, about	8

This classification of simple and complicated may be wholly factitious and due to incomplete notes of the "simple" cases. The complications mentioned are: great pain with attack; weakness, permanent or transient, as well as paralysis; contracture (temporary).

It is quite likely that similar complications were present in other cases, but they are not mentioned. The weakness noted, either persisted after the paralysis had disappeared, or alternated with it. The line cannot be drawn between certain degrees of weakness and incomplete paralysis.

Before going further we may define a typical case as one in which local spasms or convulsions are immediately followed by marked weakness or paralysis which is distinctly temporary. If the case merges into or is complicated at any stage by apparently permanent paralysis it is hardly typical.

Stopping now for a brief analysis of atypical and irrelevant cases, we find that out of the eighteen we may exclude the two of Adler as probably permanent and due to trauma of the musculo-spiral nerve during convulsion. Of the remaining sixteen, analysis is as follows:

So-called inhibitory paralysis or paralysis without convulsions in	8
Strong probability that paralysis was permanent in sensory paralysis instead of motor in	5
Sensory paralysis instead of motor in	1
Contracture instead of paralysis	1
Paralysis occurred only when attack of convulsions was aborted by constriction of limb	1
Total	16

It is very difficult of course to determine whether many of the above conditions may be regarded as "equivalents" of ordinary post-epileptic motor paralysis. Doubt-

less, authorities would differ as to admitting the paralyses not preceded by convulsions. Jackson says they have nothing to do with the case, while Gowers regards them as a kind of equivalent.

As to whether or not temporary exhaustion can be substituted by any of the numerous so-called equivalents, we have no adequate evidence, and we probably have no more justification for assuming these equivalents in temporary paralysis, than we have in substituting the so-called "psychical" equivalents for true epileptic fits. The whole subject of equivalents in epilepsy is certainly in urgent need of revision.

The undoubted typical cases are those of Blaud, Bravais, Pereboom, Charcot, Grasset, Echeverria (6), Hughes, Todd (10 cases, all but cases 10 and 11 of our notes), Dutil (2 cases, 2 and 4), Féré, Robertson (2 cases), Russell, Jackson (3 undoubted cases, Nos. 1, 5 and 6), Heurot, Griesinger, Hitzig, Charcot and Pitres, Gowers (2 cases, 1 and 4). Total, 38.

The cases of "inhibitory" paralysis are those of Pavy, Fournier, Todd (Case 11), Féré, Russell (2 cases), Gowers (2 cases). Total, 8.

The cases in which it appears impossible to determine whether or not exhaustion played any rôle are those of Sturges, Bourneville, Comby, Dutil (Case 1), Robertson (Case 2). Total, 5.

Case of sensory paralysis only—Todd (No. 10).

Case of contracture instead of paralysis—Dutil (No. 3).

Case in which paralysis appeared only when attack was aborted, also of Dutil (No. 5).

Analysis shows that in the great majority of typical cases the attacks agree as to symptoms. The convulsions as a rule are hemiplegic. Reports do not always record

whether or not the hemiplegia includes the face. Sometimes it is stated that the attacks begin in one or other extremity, but a large proportion of the authors are silent on this point. In thirty-two cases, sooner or later there were hemiplegic convulsions and paralysis. Echeverria mentions one case of paraplegia, while Féré records an attack of temporary paraplegia in a patient subject to hemiplegic attacks. In one of Todd's cases and also in one of Gowers' the paralysis was confined to the arm, while in Hitzig's case it was confined to the face and tongue. Authorities state that the arm is more frequently and severely affected than the leg and that the arm often is completely paralyzed, while the leg is only partly paralyzed. On looking over my own cases it would seem that the preponderance of the arm over the leg paralysis is not very large.

While the typical cases are tolerably uniform, there were quite a number of complications, such as attacks of grand mal, painful spasm or paralysis, contractures (temporary), and persistent or transient weakness. In several cases an initial tonic spasm is described as occurring before the clonic movements. In several other cases the earliest fit did not result in any weakness or paralysis, while subsequent seizures were followed by loss of power.

The duration of the paralysis varies extremely and does not appear to bear any special relation to the severity of the convulsions.

In several cases there were bilateral convulsions although one side always greatly predominated; and Robertson records a unique case of alternating convulsions and paralysis, the two sides being alternately involved.

In Charcot's case, clonic spasm appeared to be followed by a tonic condition (in the leg).

Of the typical cases of exhaustion-paralysis, the etiology is not less obscure than in epilepsy in general. From reliable data we are able to give some definite cause in only thirteen out of thirty-eight cases. The definite factors are as follows:

Syphilis	4
Trauma	3
Tubercle	1
Hydatid	1
Puerperal (uræmic?)	1
"Cortical lesion"	1
Chronic brain-disease, atrophy, focal softening	2
	13

Of the eight cases of inhibitory paralysis, only three have any definite cause:

Syphilis	1
Sunstroke	1
Chronic brain-disease (atrophy)	1
	3

Of the five doubtful cases, three occurred in children, and one is recorded as having been due to hypertrophic sclerosis. Upon reviewing these five doubtful cases, it seems that possibly one case (Dutil's) could be included among the typical class, while the other four had better be left out of consideration entirely.

In the Todd's case (No. 10) with repeated attacks of temporary sensory paralysis, and also in Dutil's case (No. 3) of repeated attacks of temporary painful contracture following spasm, there is no definite cause ascertainable.

In Dutil's case (No. 6) of paralysis which would supervene whenever constriction was applied to the limb, we have the only definite causal element anywhere upon record external to the central nervous system.

Bravais'[40] thesis is not to be found in this country. The only case of his accessible is the one cited by Dutil, in which an attack of epilepsy was followed by hemiplegia which was general. This disappeared of itself. Three weeks after the attack both sides were found normal.

Although the etiology of the epilepsies in which exhaustion-paralysis appears is many times founded upon a definite organic lesion in the brain, yet the mere fact that some cases show no organic or even cytological changes by the aid of the microscope, makes us regard the etiological factors given in this chapter only in the light of determining factors to the exhaustion phenomenon.

In looking over exhaustion cases in literature, the author has found it impossible to determine whether a given case was really in the beginning due to a unilateral lesion of the brain, and, therefore, what is known as a partial epilepsy, or, whether the case was of that rarer form of epilepsy in which exhaustion-paralysis appears without any cause except functional exhaustion in the fit either local or general.

Todd's[41] Cases.

Unilateral Epilepsy and Hemiparesis.

1.—Male, æt. 10. February, 1853. First fit four months previously. No apparent cause. First attack bilateral, but all subsequent fits right-sided. After each fit right side paralyzed, muscles relaxed. Motor paralysis only,

which is incomplete; slight power left. Fits last four or five minutes, with very short coma. Interval between fits varies. After long periods of freedom, as many as eight daily.

2.—Male, æt. 34. March, 1850. Fits for about fourteen years. Begin with twitching in left fingers. Come on quickly. Left arm and leg and left side of face paralyzed. Fits last ten minutes. Patient never entirely recovered from weakness between paroxysms. Always worse after each fit. Also minor fits without loss of consciousness. These minor seizures did not increase the weakness.

3.—Male, æt. 26. December, 1851. Fits for eight years, at irregular intervals. Quick recovery but right side always remains paralyzed or weak. Paralysis never complete. This case was probably syphilitic; but the notes are incomplete.

4.—Female, æt. 29. May, 1850. First fit January, 1850. Next one in March. Paralysis of left arm, leg, side face, and left upper lid. Paralysis lasted only a half hour.

Notes in this case are incomplete.

Paresis of Left Upper Extremity with Pain.

5.—Female, æt. 24. April, 1850. Fits for three years. Coma for half an hour. When roused, left arm paralyzed; disappeared on next day. After every fit there was paralysis with severe pain of the left arm. Both subsided in one or two days.

Unilateral Convulsions and Hemiparesis.

6.—Male, æt. 58. October, 1851. First fit February, 1851. Subsequent fits began with numbness and twitching of right great toe, gradually running up leg, side and arm, followed by convulsions. Attack would often end then; it could also be aborted by rubbing or by ligature. When the fit could not be prevented, the right-sided convulsions

were followed by paralysis of right arm, leg, and side of face. Even after four or five weeks the arm was weak and grasp feeble. Leg weak; patient limped.

7.—Female, æt. 63. September, 1853. First fit occurred in November, 1852. Patient came out with left-sided paralysis, but soon recovered. Sensation of numbness and coldness persisted. The next fit occurred in July; coma for three or four hours; decided paralysis of left side; could not protrude tongue straight. Unable to walk for three or four weeks. Another fit occurred in September; followed by coma for one hour; and almost complete paralysis of left arm and leg. Within three days there was gradual recovery of sensation and motion, but numbness and coldness persisted.

This fit was not typical of epilepsy.

8.—Male. First fit in April, 1852, and since then recurrence every two to six weeks. The fits begin with giddiness and last two to three minutes; leaving patient with temporary left-sided paralysis, most pronounced in leg. Paralysis disappeared in fifteen or twenty minutes, and next day recovery was complete.

9.—Male, æt. 25. Recent syphilis. First fit September, 1853. Was followed by left hemiplegia. Four days later still had paralysis and relaxed muscles; tongue deviated to left; left side face paralyzed; pupils dilated. Paralysis lasted but a few days.

Sensory Paralysis Only.

10.—June, 1854. Patient of Dr. Spender. Male, æt. 42. First fit winter of 1854. Coma lasted one hour. Complete anæsthesia left side. Motor power unimpaired. Speech affected. Reflexes not abolished. Following summer had similar seizures.

Apoplectiform Epilepsy; (Inhibitory Paralysis?).

11.—Female, æt. 38. January, 1846. First fit in April, 1844. More like apoplexy than epilepsy. Comatose twenty-four hours, followed by left hemiplegia and

aphasia. Recovery slow and partial. General paralysis set in, followed by death in three months. Died comatose and convulsed.

Autopsy.—Large Pacchionian bodies; arachnoid very opaque. Hemispheres flabby, sulci wide, and occupied by considerable subarachnoid fluid. Atheromatous spots in arteries of base, especially in the basilar and middle cerebral. Right lateral ventricle dilated, holding 1 oz. fluid. Foramen of Monro large; lateral ventricle not much increased in size. Septum lucidum very thin and firm, right corpus striatum smaller and less prominent, also softer than left. Slight softening of hemispheric substance. Gray matter pale, especially on right side. The effusion into right ventricle was due to shrinking only.

Incomplete Epilepsy with Hemiparesis.

12.—Male, 39. November, 1852. First fit December, 1851. Minor attack followed by loss of use of right arm and leg, sight and speech. Recovered in ten or twelve weeks and resumed work. A precisely similar attack occurred in April, 1852. Recovery from paralysis was incomplete. Milder attack November, 1852, followed by increase of paralysis, and speech disturbance. After two more attacks death occurred, preceded by symptoms of diminished vigor of mind and body.

Autopsy.—Abundant subarachnoid fluid; opacity of arachnoid worse on left side. Convolutions wasted, sulci large. Hemispheres, especially the left, softened and soaked with fluid. Left thalamus larger and more flaccid. Corpus striatum and rest of brain normal except less firm.

Robertson's[42] Cases.

Repeated Seizures with Paralysis after Each.

1—Alternate right and left unilateral convulsions and hemiplegia; trephining; left hernia cerebri; no loss of speech; death after six months; autopsy.

Male, æt. 43; seen August, 1868; necrosis of skull,

mostly on left side, from injury. General condition good. On September 3d had four left-sided convulsive seizures of ten minutes' duration each, followed by complete loss of power of left arm and leg. Sensibility much impaired. Convulsions reappeared in evening. Right half of body not involved except abdominal muscles and sterno-mastoid. Head lay on left side, left divergence of eyes, left pupil dilated. After awhile the right leg began to act synchronously with left, while the right arm and right side of face followed. The convulsions were now general throughout the body, the right side predominating. Patient was now trephined. Recovery from paresis was rapid. (Evidently it is not easy to say whether the recovery was spontaneous or due to operation).

On October 16th, right arm and leg convulsed, beginning with a tremor of the hand. Convulsions lasted 2½ hours and the right extremities were found partially paralyzed, the arm being the worst. On November 1st, a right-sided seizure. After this no more convulsions. Hernia cerebri developed at the sight of trephining. An abscess formed and patient died in coma. He had nearly recovered the use of the right side.

Autopsy.—The only lesion was a deep abscess-cavity in the parietal lobe corresponding to the bone lesion, with softening of immediate vicinity. Texture of the mass of brain healthy. There were two other regions where a slight degree of circumscribed softening was noted, viz.: surface of the right post-parietal lobe and at another point over the parietal lobe where the dura was adherent.

Case of Paralysis which may or may not have been Due to Exhaustion.

2.—Female, æt. 59; on January 14, 1869, right-sided convulsions, beginning in the leg and subsequently involving arm and pectoral muscles; still later the right side of the face and abdomen were involved. During the more severe attacks the left abdominal and sterno-mastoid muscles participated. Convulsions lasted 1½ hours and

the right side was found paralyzed. Patient died a few days later. An autopsy was not allowed.

This was probably a case of true apoplexy supervening, possibly, after epilepsy.

Cases 3 and 4 were examples of unilateral convulsions, but the paralysis was not of the temporary exhaustion variety.

Convulsions Followed by Total Paralysis of Very Brief Duration.

The following case, which Robertson describes in a foot-note, shows how very transitory may be the paresis which follows epilepsy :

5.—An elderly person attending worship in a church, where Dr. Robertson was also present, was seized with a fit. By the time Dr. Robertson had reached his side (about one minute after the beginning of the seizure) he was no longer convulsed but simply unconscious and breathing calmly. He was carried out into the session-house and examined. His right arm fell heavily at his side when raised a few times; the right leg also seemed feebler than the other, the left leg and arm were in no way affected. In less than ten minutes he was using the apparently palsied arm with as much freedom as the other. In about half an hour he had fully recovered consciousness and then said that he had been an epileptic for thirty years and that the fits affected the right side especially.

Jackson's[43] Cases.

Partial Epilepsy followed by Hemiparesis.

1.—Male, æt. 48. Fit began with prickling of fingers, followed by tonic spasm of fingers with slight tremor. Fingers incurved toward the palm. Spasms soon passed from tonic to clonic. Possible doubt as to existence of a tonic stage. Later the fingers became flexed, and the hand

pronated. Pain in the hand was marked. There was pain and spasmodic motion in right knee. Absence of face twitching and speech derangements; consciousness retained. After movements ceased, right arm and forearm were nearly powerless. Weakness of the right leg also interfered with walking.

2.—Occurred 1862. When fits began, index finger pointed straight out; thumb approximated. Other fingers curved in with convulsive twitchings. Notes in this case were incomplete.

3.—Began with sudden sensations in right hand; index and thumb drawn up together. Convulsion ran up from hand to arm, thence to face with loss of speech. Consciousness not disturbed. Attack passed down to side and leg and out at toe. This case died.

Autopsy.—No local gross lesions. Atrophy of both sides of brain.

In a similar case Jackson found a mass of tubercle in the back part of the third right frontal convolution. In this case convulsion began in thumb.

Partial Epilepsy; Painful and Nocturnal Inception.

4.—Patient fell asleep in a chair. Waked with great pain in second and third fingers. Then thumb stiffened and convulsions began, at first weak, then stronger, extending up the arm to the face.

Convulsion Followed by Absolute Paralysis; Consciousness Retained.

5.—Male, æt. 48. December, 1868. Attacks began by twitching of right thumb and index finger. Other fingers followed; fingers flexed in a curve except the index which was straight. Both index and thumb were flexed on hand and mutually approximated. Later the whole arm twitched without locomotive effect. Two minutes after fingers twitched, face was involved. Right eye closed, right cheek drawn up, jaws closed, mouth drawn to right,

buccal opening ovoid with wider end to right. Ocular apertures, especially the right, narrowed. Both sides of the forehead wrinkled upward. No deviation of head or eyes. Leg not involved. No aphasia,—could talk at any time during fit, which ceased suddenly. The arm, already weak before the fit, was completely paralyzed; was limp and fell forward when he stooped. Two weeks later (when discharged from hospital) was not completely cured. Attack lasted ten minutes. Bystanders said that the arms were strongly convulsed.

Temporary Hemiplegia after Localized Epileptiform Convulsions.

6.—Female, æt. 42. June, 1872. Syphilitic. Double optic neuritis. Has had many fits, beginning by right-sided spasms. Conscious throughout. Right hemiplegia followed. Some aphasia, but tongue not paralyzed.

GOWERS'[44] CASES.

Painful Spasm followed by Extreme Weakness.

1.—Male, æt. 20. Fits for ten years. Commence with pain in front of right shoulder and run down arm. No spasm. Twitching at right angle of mouth. Mumbling speech. Whole attack only momentary. Pain in arm replaced by spasm. From time pain began, arm became weak and could hardly be raised. Weakness lasted a quarter hour.

Weakness not Preceded by Spasm (Inhibitory).

2.—Male, æt. 25. Fits for nineteen years. Right-sided; no paralysis with these major attacks. Also has minor seizures, characterized by mumbling speech coming on suddenly, use of wrong words, etc. There was weakness of right arm, not preceded by spasm.

3.—Female, æt. 40. Slight right-sided weakness and hemianæsthesia. Attacks begin with tickling of right ear followed by pain running down side of leg and foot; after

reaching toes it extends up leg and side and reaches the arm, hand and tongue. In half an hour she mumbles and cannot speak. There is no spasm, but weakness sets in when sensation is perceived. Patient is unable to stand and can hardly raise her arm.

Complete Hemiplegia Ceasing Spontaneously.

4.—On three occasions, extending over about ten days each, a patient had more than one hundred unilateral fits daily. Complete hemiplegia and aphasia followed, which disappeared spontaneously after fits ceased.

CHARCOT-PITRES'[45] CASES.

Dutil in his article cites four cases from the article published by these authors on localization, etc., as examples of exhaustion-paralysis.

Convulsions Followed by Various Forms of Paresis.

1.—Male, æt. 28. November 6, 1876, was taken suddenly with a convulsive attack which began in left hand with violent contraction of fingers; little by little these convulsive movements extended to the forearm and arm and consciousness was then lost. Two days later a new and similar attack began and left a paresis of the left arm and leg. Face normal and sensibility preserved. In other attacks, the left arm was most involved. Death resulted (no particulars) on January 21, 1877.

Autopsy.—Tuberculous mass buried in the gray matter of the right ascending frontal convolution at the level of the middle third. Small recent focus at right side of protuberance. (This case was originally published by Heurot[55]).

2.—Male, æt. 41, taken suddenly with a clonic convulsion of the right leg, only momentary. Recurred repeatedly several times daily. One month later there was extension to right arm. Voluntary movements of the right leg brought on the spasms which extended to

the whole right side. Head and eyes were turned to the right. Consciousness lost. In the intervals the right arm and leg were completely paralyzed. Face very slightly affected. (This case was originally published by Griesinger).

Autopsy—Hydatid cyst, 4 cm. broad, 4.3 cm. long containing six small cysts. Was seated on the left side of the brain in such manner that its anterior extremity reached to the level of a vertical line drawn through the auditory meatus. Five other detached cysts of the size of a kidney bean were found on the surface of the left hemisphere.

3.—Soldier, injured in head December 10, 1870. Wound appeared to be doing well when on the 4th of February clonic convulsions set in, chiefly of the left side of the face. Consciousness retained. This attack was followed by paresis of the left facial nerve and left side of tongue. Other attacks followed and the facial paresis seemed nearly gone, when there was an epileptic attack involving the left pectoralis major and upper abdominal muscles of both sides. Patient died February 10th.

Autopsy.—Purulent focus at site of injury. Its upper border was 6½ cm. from the median line; lower border 2 cm. below the fissure of Sylvius; it lay in front of the fissure of Rolando, between the latter and the prefrontal fissure, within the ascending frontal. Texture of brain normal except softening in the immediate vicinity of the abscess. There were numerous hemorrhagic points scattered through the cerebral substance, most numerous on the left side. (This case was also published by Hitzig[54]).

4.—(No. 34). Female, æt. 52. October 9, 1876. Had convulsions a year before with loss of consciousness and was ill for several days afterward; for about a month incomplete paralysis on left side. Mental processes enfeebled. On October 27th seized with persistent epileptiform convulsions without regaining consciousness. Death followed next day. Status epilepticus and cyanosis.

Autopsy.—Lesions were too numerous and varied to

throw much light on the case. Left cerebellar lobe was much softened. In the right cerebral cortex, there were four old superficial foci of softening—two of these were on the frontal convolution, one was on the sphenoid and one on the ascending frontal convolution.

Chapter VI.

CASES OF EXHAUSTION-PARALYSIS IN EPILEPSY CITED IN LITERATURE—*Continued.*

Féré's[46] Cases.

Cases of Exhaustion Paralysis in Epilepsy.

1.—Male, 30; May 29, 1889. First fit when 7 years old, nocturnal. Probably grand mal. Another fit when aged 10, partial. After that, habitual attacks, two or three per year. As a rule the right side only was involved. During 1879 he awoke on two occasions with paralysis. On the first occasion, right side paralyzed for one day only; on the second occasion he had general paralysis with aphasia, paralysis of sphincters, etc. This lasted three or four weeks and disappeared all at once.

This case was studied at the Bicêtre in 1880. Convulsive attacks involved arm and leg of same side (right) and then became general. The arm was frequently found to be very weak after seizures. Aphasia was never entirely recovered from after attack of complete paralysis.

2.—Male, 42. When 28 had his first seizure after a sunstroke; grand mal. Ten days later had a series of twelve attacks in one day. Then for the next ten years an average of two attacks a month, usually when he awoke in the morning. For past four years has been under the influence of bromides. No attacks of convulsions, but instead, about once in two months, apoplectiform attacks without loss of consciousness. Patient falls and is helpless for several moments, quickly regaining his power.

By some this case would be called "inhibitory" paralysis; as no apparent convulsion preceded the paralysis.

DUTIL'S[47] CASES.

Post-epileptic Weakness Becoming Permanent.

1.—Male, æt. 52; July 13, 1882; fits for five years. First attack began in right great toe. Foot and leg shaken by convulsions involving side of body as high up as arms. Complete epileptic seizure. When he came to himself, in half an hour, he had to be supported home. Since then he has had many attacks, complete or incomplete. In 1879 developed first stage of phthisis. Attacks now began to be followed by weakness which persisted four or five hours. They became more frequent and left patient in state of hemiparesis. Besides paresis of the right arm and leg, there was difficulty of speech. Left arm much stronger; left leg better than right.

The character of the attacks is illustrated by the fit which occurred on August 21st. This attack was accompanied by a severe convulsion, sensitive aura from right great toe, and finally to complete right-sided convulsion. There were three convulsions in rapid succession, the last two with loss of consciousness. Patient much depressed, speech hesitating. No deviation of features, tongue, etc. Marked, incomplete paralysis of right side. About two hours after attack, no control of leg, which dropped back whenever raised, in spite of patient's efforts. Four or five hours later the patient was able to feed himself. Right side always a little weaker.

Partial Epilepsy Followed by Temporary Paresis.

2.—Female, 20. Convulsion of entire right side. Paresis of convulsed limbs after an attack. Spontaneous disappearance in a few hours. Always had nystagmus. Admitted March 12, 1882. In August, 1881, experienced a sudden weakness of left side. Formication in left little finger. Tonic spasm of left side, beginning in left arm and succeeded by violent convulsions. No loss of consciousness. In a second attack on same day, consciousness lost. Next day, some paralysis. During April,

1882, an attack began in left foot with formication. Convulsion ran up leg, thigh, arm and corresponding side of face.

Since then, attacks have succeeded at first irregularly, but later every eight days. Between paroxysms, he is very awkward with left hand,—often dropping objects. Left leg is also weak, so that the day following paroxysm, she keeps seated.

A characteristic attack began at 5 P. M. in left little finger. Loss of consciousness. First attack lasted fifteen minutes and was followed by three others a few moments apart. All over by 5.30. Profound depression. At 6.25 left arm very weak; could be raised to mouth very slowly and irregularly. Left leg also weak. Had to be supported to walk. On following day better, but still weak. Could sew on second day after attack.

Epilepsy Followed by Aphasia or Contracture Instead of Paresis.

3.—Epilepsy with motor aura and post-epileptic transitory contracture. Male, æt. 20; June 5, 1875. First attack April, 1875. Painful spasm in right arm beginning at wrist and running up forearm and arm, preceded by a motor aura. Lost consciousness. Strong contracture of fingers and toes of right side, on account of which patient kept his bed for two days. On June 4th, violent convulsion; unconscious half an hour. Then violent spasms of right hand, running up arm and down right leg, but not followed by convulsions. On the succeeding day similar spasms were followed by convulsions, and on the sixth day right hand was in a state of painful contracture. On left side of the body a mild degree of contracture. In this case there was no paresis but temporary contracture.

4.—Choreiform convulsions (probably epileptic) of left arm and left half of face. Later on, partial epilepsy of same localities. Post-epileptic paralysis and contractures followed.

Male, æt. 39; February 19, 1882. Always well until

1880. One morning waked up ill and fell while dressing. Intense headache was followed by rhythmic contractions of left arm and left side of face. Lasted five weeks and disappeared leaving the arm weak. Early in April, 1881, regular epileptic seizures limited at first to left arm and left side of face. Within following ten months there were six attacks, local at first and then general. After these attacks, there was a certain awkwardness and stammering, which was transitory. During April there were three attacks, which began in left leg and ascended to the hip, left arm, left side of the chest, with loss of consciousness. Attacks were followed by marked contracture of the left leg.

During the next four months there were no general attacks, but local spasms and pain (in left leg) followed by weakness. In August, 1881, a general attack. Three and a half hours later the convulsive parts were in a state of paresis preventing work.

5.—Partial epilepsy which began in left leg. Paralysis of left arm and transitory aphasia in consequence of aborting an attack artificially.

Male, 38. Had always been well. First attack at night, January 15, 1872; a second in 1880 and a third in 1881. From then on had two per month. Motor aura in foot. Convulsions spread to thigh. Attacks could at times be aborted by constricting leg. On one occasion aborting the attack caused paralysis of the arm, complete at first, gradually disappearing. Paralysis most marked in extensors. Some aphasia co-existed. Next day he was entirely well. In this case paralysis appeared only upon aborting a convulsive attack.

Dutil appears to regard aphasia, paralysis and contracture as motor equivalents. He says that in some cases we have transient paralysis, others have aphasia and others again contracture.

Russell's[48] Cases.

Three Cases of Hemiplegic Epilepsy.

1.—Male, æt. 34. Had numerous fits of rapid clonic spasms of the whole right side of the body. These attacks were varied with seizures of ordinary major epilepsy in which both sides of body participated. The unilateral clonic spasms were followed by paresis of the right arm and leg (the affected areas), including the shoulder. After four or five days the paralysis would gradually lessen. Latterly the period of recovery has been more prolonged, especially in the leg; three weeks have elapsed before it entirely recovered its power.

2.—Female, æt. 30. Subject to sudden attacks of complete loss of power in the left arm exclusively, of brief duration. This paralysis is always associated with sharp pain and numbness in the arm. There is no evidence of any convulsive movements. These attacks last from one to three hours; once only the duration was twenty-four hours. The arm has become permanently enfeebled, but there are no disturbances of sensation. During one seizure only patient was unconscious throughout a whole night.

3.—Male, æt. 44. For the past six months has had fits as follows: There is a painful paralysis with numbness, which began in the abdominal muscles and extended from thence to the lower and upper extremities. Duration of attacks, about ten minutes. In the course of twenty minutes regains entire use of limbs.

For past six weeks has also had fits more characteristic of major epilepsy, but the left side is more severely involved. Since this time the minor attacks have been worse.

Mercier's[49] Case.

Tetanoid (?) Epilepsy with Exhaustion-Paralysis; Serial Attacks like Status Epilepticus.

1.—Female, æt. 29, first fit in the ninth year; Fits at irregular intervals ever since. Prolonged aura, lasting one or two hours, symptoms very numerous and peculiar—

among others, earthy pallor, defective articulation, strabismus, extreme languor. When strabismus appears, the muscles of arm and hand are found to be rigid. This state of rigidity persists throughout the epileptic attack. Superposed upon this rigidity are other symptoms which are in some respects like clonic convulsions, since they are intermittent, but the tonic element is evident even here, the movements being slow, flowing and continuous.

The rigidity which persists between the convulsions and for hours after they are over consists of opisthotonus of the trunk, legs and feet extended, arms at side or in front of trunk, forearms strongly pronated and hands clinched, spasm of platysma, rapid temor of whole muscular system. The convulsions always begin at the periphery and are bilateral with predominance of right side. Hands, feet and face are simultaneously affected at the onset. The right side is usually paralyzed after the fit; on one occasion the left side predominated in the convulsions and then the left side was paralyzed. Several days elapsed before consciousness was fully restored. After a prolonged attack, October 1 and 5, the right leg remained perfectly paralyzed and anæsthetic. On October 11, after another attack, the right arm remained paralyzed, anæsthetic and rigid for six months.

In concluding Mercier dwells upon the essential *tonic* character of the whole affection. There are also many peculiarities of speech described in connection with this patient.*

Woodbury's[50] Case.

Exhaustion-Paralysis Following Infantile Spasms.

Child, aged 21 months; general convulsions of unknown origin. After first attack right side seemed to be paralyzed. Convulsions were then confined to left side, and

* For description of a similar case in which tetanoid seizures formed a status period, see a case reported by the writer in the *American Journal of Insanity*, April, 1899.

after repeated attacks the left side became paralyzed. The right hemiplegia disappeared very soon after it came on, but in the left side power returned only after a week of active treatment. The right hemiplegia was somewhat obscure in nature, but Woodbury regarded the left hemiplegia as due purely to exhaustion.

Blaud's[51] Case.

This is one of the first cases of exhaustion-paralysis on record. Child of seven years; convulsions, followed by right hemiplegia. Spontaneous disappearance of paralyses in two hours. Eleven days later convulsions followed by paralysis of right arm. Autopsy revealed a cortical lesion of left hemisphere.

Pereboom's[52] Case.

This is a case of temporary exhaustion-paralysis following eclamptic convulsions in childbed.

Charcot's[53] Case.

1.—Male, æt. 42. When first noticed his right lower extremity was violently convulsed. After a few seconds the whole limb became rigid and consciousness was lost. Came to himself in an hour. Two months later had another attack. This time the leg was somewhat enfeebled afterwards; the arm was also involved. Two months later a third attack; it was then demonstrated that the convulsion became general, involving first the leg, then the arm, and then the body as a whole, but preponderating on the right side. When seen by Charcot the right upper and lower extremities were somewhat weak. Hemiplegic epilepsy is one of the most frequent symptoms of syphilis. This case quickly responded to specific treatment.

Charcot gives other cases where paralysis was probably present, but gives no details.

Grasset's[54] Case.

Case from notes furnished by Dr. Caizergue. Female, æt. 55. First convulsion not followed by paralysis. Second attack left the left side paralyzed for twenty-four hours. Third attack was followed by coma for two days. Three days after attack left side weak. After she came under observation, she had seven attacks in succession with paralysis of left arm and leg, most marked in the arm. Left side of face paralyzed. Soon afterwards these paralytic symptoms appeared to have become permanent and death set in.

Autopsy.—Diffuse meningo-encephalitis. Focus of red softening at the foot of the two first frontal convolutions. Death was due to fracture of the skull caused by a fall during the fifth seizure. Death took place in coma and high temperature.

Pavy's[55] Case.

Epileptiform fits followed by aphasia and partial hemiplegia.

Male, æt. 39. October 22, 1880. First fit July, 1879. No loss of consciousness. Aphasia and sudden loss of power in limbs. Was abed several days and confined to house for weeks. These apoplectiform attacks were repeated a number of times. As a rule patient would be left helpless for four or five hours and would have aphasia. Power would be back on waking on next morning. In the more recent attacks there was total loss of consciousness.

The occurrence of aphasia and hemiplegia without convulsions would, according to Gowers, place this case among those of "inhibitory paralysis."

Sturges'[56] Case.

Convulsions began when patient was six months old. At first limited to left half of body and always began in left hand after they became general. Left half of body observed to be weak after each partial convulsion.

ECHEVERRIA'S[57] CASES.

Echeverria tabulates 306 cases in which epilepsy is in some way associated with paralysis. Under the latter term he includes both motor and sensory phenomena as well as aphasia, contractures, etc. Of these cases *six* are positively stated to be temporary, viz.:

Right hemiplegia	3
Left hemiplegia	2
Paraplegia	1
	6

ADLER'S[58] CASES.

Adler publishes two unique cases which he says differ from ordinary exhaustion-paralysis. There was total right-sided radial paralysis following on epileptic seizures. The paralysis was clearly peripheral in origin, but its nature was obscure. Adler finds that Gowers describes three similar cases in his work on Nervous Diseases, while Oppenheim in his work recognizes a paralysis from bruising of the musculo-spiral nerve incidental to strong contraction of the triceps. Gerulanus has endeavored to show (without special reference to epilepsy) that powerful contractions of the triceps may exceptionally disable the musculo-spiral nerve. Aside from these three citations, Adler was unable to find anything bearing on the subject.

While evidently not due to exhaustion, cases of this sort ought to be considered in this connection as post-epileptic and not due to central organic lesion.

COMBY'S[59] CASE.

Female, æt. 5. Seen June 26, 1898. Had had repeated attacks of nocturnal epilepsy since November 23, 1897, as many as eight or ten fits in succession. When first seen there was left hemiplegia involving limbs and face. The paralysis was incomplete; child could move about in bed

but not stand. She continued to have left-sided convulsions. On account of persistence of hemiplegia, craniotomy was performed. No morbid focus found on the cortex and the wound was closed. Child never recovered from operation and died comatose.

Autopsy.—Everything absolutely negative as to foci. No tumor, abscess or other circumscribed lesion. The right hemisphere was thought to be unnaturally firm on section and a diagnosis of hypertrophic sclerosis was made. No record of microscopical examination.

Fournier's[60] Case.

1.—Male. Syphilis. First had aphasia; then temporary paralysis of right arm; then two more attacks of aphasia, the last with right facial hemiplegia and weakness of entire left side; repeated epileptiform attacks. In this case no mention is made of convulsions preceding paralysis. Coma and death.

Autopsy.—A large gumma, 3 cm. in breadth, posterior part of left frontal convolutions as well as adjacent parietal.

2.—This patient had, in addition to major attacks, convulsions of one upper extremity lasting from half a minute to several minutes. Began with a sort of spreading of the fingers, then there occurred forced flexion of forearm on arm. The entire arm was then convulsed. In the above and similar cases there is no mention of weakness or paresis.

Hughes'[61] Case.

Male, æt. 12. Traumatic (?) epilepsy. Petit mal many times daily. On one occasion coma for eight days. He also developed hemiparesis of left side, the left arm being completely paralyzed. He recovered from the paresis to a considerable extent.

Hughes says cases of post-epileptic exhaustive paralysis are common—even very frequent. In this statement he differs radically with some of the leading authorities.

Bourneville's[62] Case.

Female, æt. 23; April 1, 1862. First fit occurred at the age of thirteen, and the second a month later. Both were were associated with pain in arm. Four years later the left arm and leg had become much weakened by convulsions. Eventually, she developed permanent hemiplegia with contractures. Possibly the weakness following the earlier fits was due to exhaustion.

Prichard's[63] Case.

Male, æt. 46. Seen December 23, 1816. Of late he has been much troubled by strong convulsive jerkings of his left arm. Comes on by fits; arm violently tossed about for a minute or two; it afterwards feels benumbed and he is for a short time incapable of moving it. Several attacks daily. Each fit followed by severe headache.

Beevor's[64] Case.

Male, æt. 24. Seen December 6, 1881. First fit when six years old, followed a fall. Comparatively few attacks until present experience, when suddenly, and without apparent cause, he began to have incessant seizures (59 in one day), the right side predominating. As a result of these incessant fits he became delirious and in interparoxysmal intervals had total loss of power in right arm and leg. On December 12th unable to walk or stand. Fits subsided as suddenly as they came on. Power became normal in a few days. (This case is from service of Dr. C. Bastian).

Jackson mentions Beevor in connection with exhaustion-paralysis, but date is earlier than 1882.

Although we have carefully collected these illustrative cases from the principal writers upon the subject, we are fully aware that the collection is not complete. Exhaustive cases appear many times in general literature, and, by the discerning eye, may often be detected when least

suspected. It is unnecessary, even if not impossible, for us to search out all the isolated reports of cases. Russel[65] in two papers entitled, "Cases illustrating the influence of exhaustion of the spinal cord in producing paraplegia," describes several cases of exhaustion-paralysis due to over-exertion, but these fall into the category of exhaustion-paralysis independent of epilepsy, already considered in Chapter III. Two cases reported by Féré, one with paralysis from wielding the hammer, and another from excessive piano practice, also belong to the same class.

Synopsis of Thirteen Autopsies.

(For details see cases).

1.—*Blaud.* Temporary paralysis following convulsions; phenomena repeated with death. Autopsy: cortical lesion left hemisphere.

2.—*Todd.* Inhibitory or apoplectiform attacks, without convulsions. Autopsy: atrophy and softening.

3.—*Todd.* Temporary paralysis following convulsions, merging into fatal cerebral disease. Autopsy: atrophy and softening.

4.—*Robertson.* Repeated attacks of temporary paralysis. Trephining; death from operation. Autopsy: only lesion, abscess cavity (abscess due to necrosis from injury).

5.—*Fournier.* Temporary paralysis alternating with convulsions. Autopsy: large gumma, left frontal convolution.

6.—*Heurot* (Charcot and Pitres). Temporary paralysis after convulsions. Autopsy: tubercular focus, right ascending frontal convolution.

7.—*Hitzig.* Temporary paralysis (facial) after convulsions. Previous recent injury to head. Autopsy: purulent focus at sight of injury.

8.—*Griesinger.* Temporary paralysis after convulsions. Autopsy: hydatid cyst surface of left hemisphere.

9.—*Grasset.* Temporary paralysis after convulsions. Fracture of skull; death. Autopsy: diffuse meningo-encephalitis; foci of softening.

10.—*Charcot et Pitres.* Temporary paralysis after convulsions. Death in status epilepticus. Failure of mental power preceding. Autopsy: numerous foci of softening.

11.—*Comby.* Child; temporary (?) paralysis which became confirmed. Craniotomy. Death. Autopsy: no focal disease; diagnosis of hypertrophic sclerosis.

12.—*Jackson.* Typical; no mention of paresis, which was doubtless present. Autopsy: bilateral atrophy.

13.—*Jackson.* Similar to preceding. Autopsy: tubercular focus, third right frontal.

Comparing the table of autopsies with the preliminary analysis of cases, we find eight of the thirteen occurred in cases of typical exhaustion-paralysis, viz.: Blaud, Todd (Case XII), Robertson, Heurot, Hitzig, Griesenger, Grasset, Charcot et Pitres. Of these eight cases the findings were

Atrophy and softening	1

Here the process appeared to be uniform throughout the brain; all the other cases were examples of focal lesion, viz.:

Abscess	2
Meningo-encephalitis with foci of softening	1
Foci of softening	1
Tubercular tumor	1
Hydatid	1
"Cortical lesion"	1
	8

It is, of course, difficult to determine the relationship between the lesions found after death and the paralysis, etc., during life; thus in the case where meningo-encephalitis was found, the lesions were probably traumatic due to a fracture sustained during a convulsion.

In two cases of autopsy where temporary paralysis was not preceded by convulsions, viz.: Todd (Case XI) and Fournier, the post mortem findings were

Atrophy and softening	1
Gumma	1

In Comby's case where the existence of temporary paralysis is not certain, but where paralysis of a permanent character was ultimately present, finding,

Hypertrophic sclerosis	1

Finally in two of Jackson's cases where paralysis or weakness is not recorded, but which should nevertheless have been present, we find

Bilateral atrophy	1
Tubercular tumor	1

Chapter VII.

THE AUTHOR'S CASES OF EPILEPTIC EXHAUSTION-PARALYSIS.

About forty cases of epilepsy exhibiting exhaustion-paralysis phenomena, forming the clinical basis of this study, have come under our own observation.

A number of uncomplicated cases have been reported. Of the nineteen cases given here, at least fourteen have no gross organic lesion and can be considered as true idiopathic cases. Idiopathic epilepsy with general convulsions and with mono- or hemi-plegia following the seizure is not common.

Classification of author's cases:

Cases of typical exhaustion-paralysis, but with general epileptic seizures. (Cases 1, 2, 3, 4, 5 and 6)	6
Cases of exhaustion-paralysis at beginning, with paralysis becoming more or less permanent. (Cases 7, 8)	2
Cases of exhaustion in spasmodic infantile hemiplegia. (Cases 9, 10, 11, 12, 13)	5
Case of exhaustion phenomenon in grand mal epileptic having an aborted attack, with unusual order of invasion. (Case 14)	1
Case of infantile palsy with exhaustion-paralysis on the normal side. (Case 15)	1
Case of exhaustion manifest in aphasia. (Case 16)	1
Case of exhaustion manifest in crossed paralysis. (Case 17)	1
Case of exhaustion in anomalous cases of epilepsy. Case 18)	1
Case of exhaustion-paralysis in status-epilepticus, with prolonged evidences of paralysis (two months)	1
	19

Cases of Typical Exhaustion-Paralysis with General Epileptic Seizures.

Case 1.—J. K., male; æt. 14. Epilepsy began at eleven with petit mal attacks which in two years became grand mal and appeared monthly in series of ten or twelve in twenty-four hours. Neurotic family history. After a series of twelve grand mal seizures he had left side paralysis for three months before complete recovery.

No evidence of weakness remained. April 7, 1898, two years after first exhaustion, he had another series of twenty-five attacks in forty-eight hours, which rendered the left side hemiplegic for four hours, and two days passed before patient regained full power. (See Text-Figure 1). No evidence of exhaustion-palsy remains. Although attacks were general, the left side participated most frequently in the seizures.

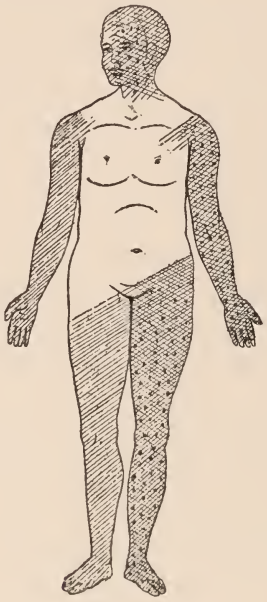


Fig. 1.

//=slight convulsion. #=severe convulsion.
::=slight paralysis.

The following case is given to show that there must be certain conditions present in the brain back of convulsive phenomena to cause even a temporary paralysis aside from exhaustion. The author urges unequal energizing of motor centres for the condition. The case at the same time illustrates that the degree of paralysis is not always dependent upon the severity of the convulsion.

CASE 2.—C. G. W., male, æt. 55; married. Epilepsy began at the age of 44; supposed cause, alcoholism. Neurotic family history. The first attacks were of grand mal, beginning on the right side. Attacks were at first preceded by an aura of twitching in right side; there are no aura at present. Knee-jerks normal.

October 23, 1898.—When the present seizure began the patient had not recovered from the effects of a previous attack of severe convulsive nature. First manifestation of the serial or imbricated attack was a slight tonic spasm of right upper extremity, the arm being flexed, the fingers clutching the clothing over the chest, the eyelids partly closed and eyeballs turned upward, head inclined in direction of onset. This condition was immediately followed by a violent clonic convulsion, first visible in left upper extremity and extending to the right lower. The convulsion in the lower limbs was not at first well marked, being confined to the calf muscles, but gradually involving the entire limb and simulating the movement of upper extremity. Face was very much congested and slightly cyanosed. After the attack had lasted about thirty minutes, during which the same general clonic character predominated. Inhalations of chloroform, given for about four minutes, moderated the convulsions, but they resumed their normal vigor when anæsthesia was withdrawn; the anæsthetic was reapplied until the attack was thoroughly controlled. Total duration of attack, thirty-four minutes. Temperature during attack, 100.6° F. One hour after attack, temperature, 99.8° F.; pulse, 80; respiration, 24. Evening temperature normal, 97.8° F. Patient rallied at 2.05 P. M. but was very much confused mentally and in an automatic state. Examination at various periods revealed no evidence of local or general paralysis. (See Text-Figure 2). Patient was quite easily aroused from stupor after first two minutes, so coma or mental hebetude did not cover paresis.

November 12th.—Patient is having many slight attacks of facial and lingual clonic muscular spasms, arythmic and irregular in character, lasting from three to eight

seconds. Consciousness is retained. Spasm involves right side of the face and right corner of mouth, but the involvement of the tongue is equally bilateral throughout. Frequently slight stertor is present after attacks and paralysis of convulsed parts follows.

The petit mal attacks when first observed, usually began with a slight tonic convulsion of the right arm which was flexed, with the forearm laid on the chest and the fingers twitching or picking at the clothing. This was generally accompanied or immediately followed by a convulsion of the muscles in the right upper maxillary region, which always continued for some time after the movements of the arm had ceased.

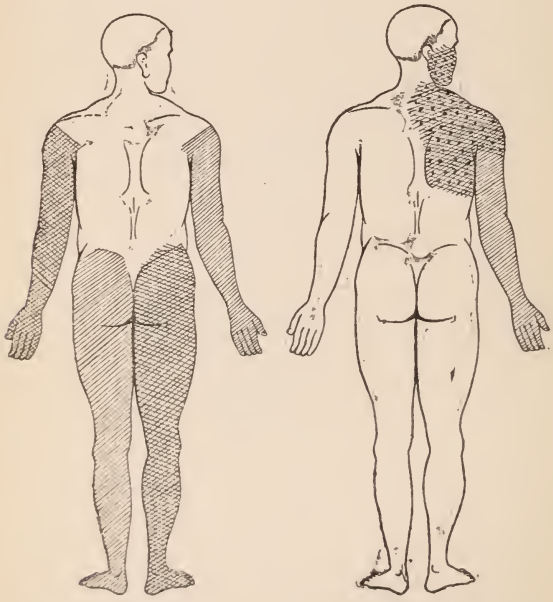


FIG. 2. FIG. 3.

//=slight convulsion. #=severe convulsion.

::=slight paralysis.

As seizures became more numerous the convulsion involved the muscles of the right face only. On three occasions it was noticed when the onset was about the right maxillary region, that the muscular agitation

extended down on the same side of the chest as far as the fifth or sixth rib and the right side was occasionally involved. Cutaneous sensation was lost for a few minutes in the chest area involved in the spasm. (See Text-Figure 3).

November 18, 1898.—During these minor attacks patient's consciousness was not perfect but might be said to be disordered. The patient was unable to preserve his equilibrium and always fell to the right side. Entire right side being convulsed. Slight paresis and diminished cutaneous sensation was found in right leg only.

November 28th.—For the past week it is noticed that after a series (20 or 30) of mild right facial and right arm spasms, the patient drags his right leg slightly in walking. There is a marked tendency for the paresis (occasionally in whole right side) to improve when seizures are infrequent. Such slight paresis lasts for several hours. Right leg is not involved in the paretic state when muscular convulsion of this extremity can be excluded. No tremor nor subjective symptoms are present. At one observation, when ten "facial spasms" alone had occurred, the elevators at the right angle of the mouth were paralyzed for several minutes. Sensation was not altered at this time.

January 13, 1899.—Patient became unconscious while sitting at dinner, and remained in this condition for two minutes, then passed into a state of rigidity which apparently affected the whole body simultaneously. The position of patient precluded the possibility of careful observation. As the rigidity relaxed, the fingers of the right hand began to twitch; this was followed by a jerking of the wrist and elbow of the right side which ceased after ten seconds. Patient continued in a general automatic condition for ten minutes. The facial muscles were not involved (see Text-Figure 4). Right side, including the face, was paretic for half an hour. Cutaneous sensation remained intact.

Some authorities might contend that an apoplectic state was present in this case, and this permanent lesion caused

a partial epilepsy. To such the following data will be instructive:

Clinical Record, March 20, 1899. Patient had two severe seizures during the preceding night, four hours apart, and at 6.15 in the morning he began to have clonic spasms which lasted until 8 A.M. He had one intermission of the convulsion at 6.45 for ten minutes. Consciousness was lost at beginning of spasm; eyes rolled upward; there was continuous stertorous breathing. *The convulsion was*

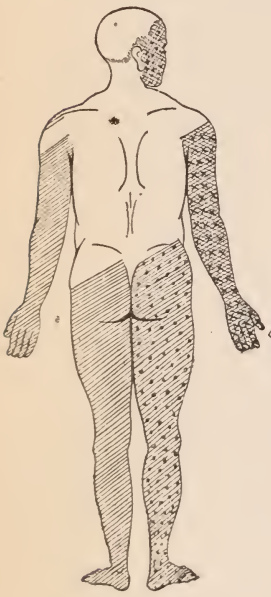


FIG. 4.

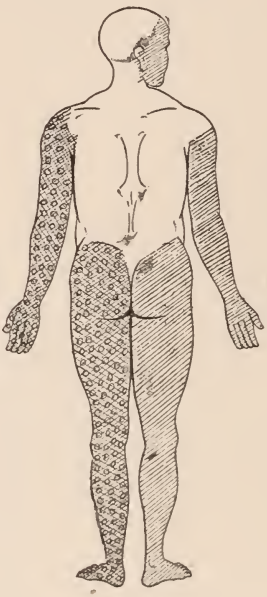


FIG. 5.

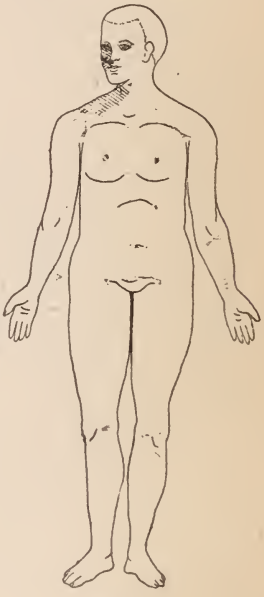


FIG. 6.

×=severe paralysis. O=contraction. Other marks as in fig. 1.

clonic on first one side of body, then the other. The head was thrown back in pillow in meningitic position and rotated every thirty seconds, first to one side, then to the other, as the convulsion predominated on one side or the other.

Profuse perspiration and cyanosis alternated from right to left with convulsion. Exhaustion was extreme; pulse

increased from 90 to 140; respiration from 18 to 34; temperature from 99.4° F. to 104° F. at 8 A. M. Conjunctivæ were hemorrhagic in spots, equally and symmetrically; pupils remained dilated. There was persistent post-paroxysmal contracture of left side for two hours after convulsion (the so-called paralytic equivalent as shown in Text-Figure 5). At 9.40 A. M. he had the usual "facial spasm" followed by deflection of tongue to right, and obliteration of naso-labial fold for two hours.

March 21.—Two attacks in forenoon, five minutes apart, consisting of tonic spasm extending left arm, and left leg was drawn up in rigidity. No other parts of body were in spasm. No after-paralysis.

March 24.—Muscular contraction of left arm with slight clonic movement on both sides of face, alternating from right to left every five seconds, left predominating; other parts of body were relaxed and did not participate in spasm. Conscious throughout, but unable to speak because of convulsion in tongue and face. Later in the day patient had a right "facial spasm," as usual, without further involvement of body. Consciousness was retained; there was right side paresis for twenty seconds after right side convulsion.

March 27.—Had general convulsion at 9.47 A. M., left arm remaining rigid at close of seizure; another general convulsion occurred at 1.50 P. M., but rigidity of arm was not so marked, and did not continue so long as before.

During the remainder of day patient had a number of slight seizures confined to twitchings around mouth, mainly on the left side, and tremor of left eyelids accompanied; during these there was an absent look in eyes, but patient was generally responsive.

March 28.—Three general convulsions occurred during forenoon; left arm rigid at close of one attack. During the afternoon mild attacks occurred about every ten minutes, confined to twitching about right mouth and tremor of right eyelids, with temporary slight paresis following for ten seconds to one minute in parts previously convulsed.

March 29.—A large number of slight attacks, the general condition being much the same as that referred to yesterday, but in addition there is a jerking of the head with deflection to right.

Only the following muscles on the right side were involved and in the order named: Omohyoid, digastric, sterno-cleido-mastoids, platysma myoides, levator labii sup. alaequenasi, zygomaticus major and minor, palpebræ superioris, orbicularis palpebrarum, depressor labii inferioris. The levator palpebræ of the left side was in clonic convulsion throughout; no other muscles on left side were involved. Right pupil markedly dilated, left much less so; consciousness retained in greater part throughout. Paresis of right side, amounting to nearly complete paralysis in parts affected, lasting for fifteen seconds to twenty seconds. Areas of the convulsions and paralysis are shown in Text-Figure 6.

March 30.—In seizures to-day, mouth is frequently held wide open, and deflected to right and upwards, the movements are more in the nature of a tremor, than of twitching as formerly.

April 1.—A series of attacks during the day, at first light, involving only the facial muscles of right side, later became general and severe, lasting from two to four minutes.

The order of involvement on the right side was: Eyelids first, next eyeball, mouth, right arm, right leg, and then the left arm and left leg; closed in same order as far as skeletal muscles were concerned; right facial disturbance ceasing simultaneously with that of the right arm. Slight paresis now manifest in whole right side.

March 20, weight 125 lb. March 22, weight 124½ lb. April 1, weight 123 lb.

March 21.—Hand grasp, right 90, left 70, in the morning; at 5 P. M., hand grasp, right 62, left 72.

March 22. Hand grasp, right 70, left 90.

April 2. Hand grasp, right 10, left 38.

Present Condition.—Patient has now had many thousand "facial spasms," and the paresis of the tongue and right face has become, in a great measure, permanent. Attention is called to Plate V, Fig. 2, which shows deflection of tongue to right and obliteration of naso-labial fold of same side seen ten minutes after a right "facial spasm." Probably actual degeneration has taken place in the motor areas supplying these parts, and thus we see a functional state so persistently continued, that at last an organic change is the result.*

Case 19 was in danger of permanent paralysis supervening upon the exhausted state and had the second status period not supervened as it did, there would undoubtedly have been a more or less complete hemiplegia. Such cases of epilepsy are in evidence frequently, proving the close relationship of epilepsy and paralytic affections.

Case 3.—G. B.; æt. 36; occupation, carpenter. All neurotic family history denied. Epilepsy began about four years ago; cause unknown. The attacks are grand mal in character and occur once in two or three weeks, generally by night but occasionally by day.

Examination of physical state of patient shows double cardiac murmur, mitral and aortic. Pulse 84. Peripheral circulation is very poor. Examination of lungs shows phthisis. Bodily condition is very poor and the skin is rough, scaly and poorly nourished. Eyes are normal except the balls, which are abnormally bulging. Speech is monotonous and nasal in character. Superficial reflexes exaggerated; the deep reflexes are markedly exaggerated; all those of the right side are greater than the left, and bilateral foot clonus is present. Motion and co-ordination

* Case C. W., Nov. 2d, 1899, has had but ten attacks for past three months and has nearly regained the use of tongue and right facial muscles. There still remains what may be termed the permanent lesion. The exhaustion element must be excluded now as the seizures have not shown perceptible exhaustion.

of the muscles are but fair on the left side but very poor on the right. Patient complains that he is unable to adjust the finer muscular movements to the necessary demands made upon them without putting forth considerable mental effort and attention (Jackson's general paralysis in epilepsy). On attempting to protrude the tongue, it is not deflected. Many stigmata of degeneration were found.

For many years prior to the onset of his epilepsy, he had periodic attacks of headache and epistaxis. During the summer months of each year patient was engaged in farm work until twenty years of age, when he began carpentry, thus following his father's trade. He has done ordinary carpentry for years but has never been considered strong and robust. No specific history or evidence of syphilis was found.

The patient's epilepsy began in the following manner: While he was at work in the factory turning at a wood lathe, he felt dizzy and confused, and upon turning to sit upon a bench near by, he staggered and would have fallen, had not a fellow workman caught him. He states that the night previous to this first attack he had been engaged as a musician at a night's entertainment in the country, and had been unable to obtain more than two hours' rest during the night, before engaging in work the following morning. He also had a very severe headache until the time of the attack. He was carried to a neighboring house and had a typical convulsion of epilepsy, and in the course of four or five minutes recovered from the attack. He found he had lost the use of the right hand and was "weak all over." Parasthesia was noticed in the right hand. (See Text-Figure 7). These motor and sensory phenomena persisted for several hours. The second attack occurred about two weeks later and was of the nature of a "sinking spell" and the right hand was "numb and useless." These two attacks were the only ones occurring during the first six months. In both instances three or four hours after the seizure, he was able to walk about the house and use his hand with as much ease as he had done before the seizures.

July 20, 1897.—No loss of consciousness takes place at present in his mild attacks, but simple numbness and noticeable loss of strength in the right hand (paralysis without convulsion). Occasionally, after severe attacks, the right leg is paralyzed but never to the same degree as his right hand, nor does it persist as long. After two of the general convulsions he has lost sensation and motion in the right side of the face, which proved temporary as all of his paralytic phenomena have. The relationship of convulsions and paralysis is seen in Text-Figure 8.

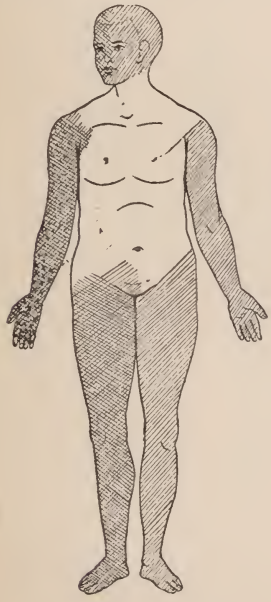


FIG. 7.

//=slight convulsion.
::=slight paralysis

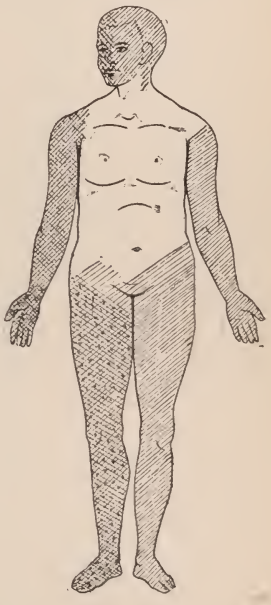


FIG. 8.

#=severe convulsion
×=severe paralysis

August 20, 1898, patient died of phthisis. He had had no convulsion for one year before his death. Nearly all of the finer muscular movements, difficult of performance while his epileptic fits were frequent, returned before he died from tuberculosis.

Case 4.—J. P., male, æt. 18. Epilepsy has existed since childhood; cause not known. Family history unknown. Attacks before admission were reported to be of grand mal character. Mental state, feeble-minded. Knee-jerks prior to admission were reported normal; on admission they were found to be exaggerated on both sides.

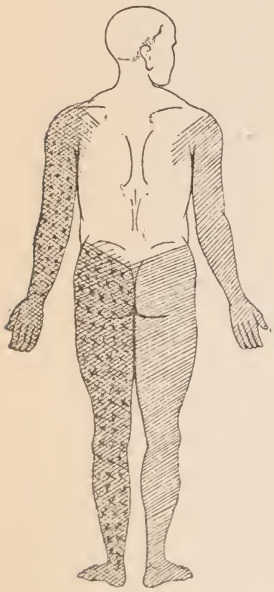


Fig. 9.

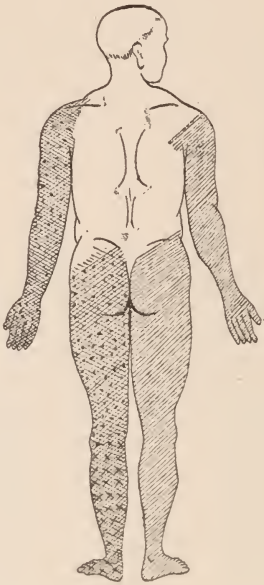


Fig. 10.

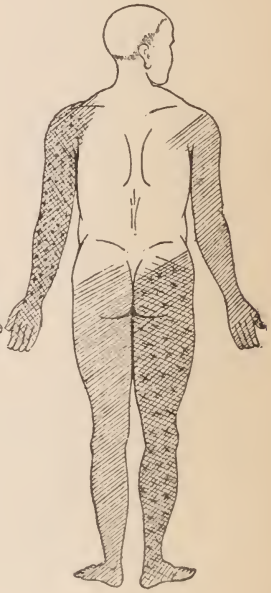


Fig. 11.

//=slight convulsion. #=severe convulsions.
::=slight paralysis. ×=severe paralysis.

After admission to the Colony, February 2, 1897, patient had many series of attacks, twenty or thirty in five or six hours. After these severe seizures, which were general but greatly predominating in severity on the left side, he suffered from a left side paralysis, which was incomplete, being most severe and persistent in the forearm and thigh, as shown in Text-Figure 9. Whenever the patient attempted to walk he dragged the left leg. This state of paralysis lasted for two hours, after which he returned to an apparently normal condition and the knee-jerk of the

left side remained exaggerated. The seizures and the paralysis were confined to the same side, in accordance with the general rule of exhaustion-paralysis.

February 16, 1897, patient had three seizures all of which were severe, and lasting from three to five minutes, general convulsions beginning and ending on the left side. The post-paroxysmal paralysis was noticeable and most marked in the left foot, shown in Text-Figure 10. The paralysis this time lasted but fifteen minutes. For the next sixteen or seventeen days the patient appeared to be as well as usual and was able to be about the house, although he had from ten to twenty severe attacks almost daily.

Upon physical examination March 10, 1897, the patient showed no effects of his paralysis other than a slight increase of the tendon reflexes of the left side as compared with those of the right. There was also a slight spastic condition in the left leg. Motion and co-ordination were almost if not quite as perfect on the left side as on the right. Since the last series of attacks (February 16th,) the patient has had many series of seizures attended and followed by temporary paralysis lasting from ten minutes to several hours before complete recovery.

During a series of ninety-eight attacks in November, 1897, covering a period of several days, twenty observations were taken for the sake of verifying convulsions and paralysis, and they were found to be as follows: The onset of tonic convulsion was in the left leg and the left arm in order given; then right leg and right arm became involved; finally generalized over entire body in severe convulsion. Reflexes of left side were lost immediately after attack while paralytic symptoms were present. They reappeared with motor control at the end of ten minutes. Right reflexes exaggerated immediately after attacks, to become equal with their opposite at end of one-half hour.

During the month of December, 1898, a series of careful observations (15) was made on different days of the series, and the following was the invariable rule for

that period: Tonic spasm in right leg and left arm, then left leg and right arm participated in the convulsion, they being mostly in clonic spasm. The order of onset of convulsion and subsequent paralysis are shown in Text-Figure 11. Immediately after attacks, right knee-jerk and right wrist-jerk were lost; after one-half hour they returned to normal. Paralysis persisted in right leg for twenty minutes and in left arm for ten minutes.

FIG. 12.

Isolated instances of crossed phenomena of convulsions and paralysis have continued to show themselves during the past three years, but have not been at all frequent or constant in the anomalous state. Paralysis always follows the general rule in this case, namely, parts most convulsed suffer most in the post-convulsive paralysis.

March 8, 1899.—Patient has had about 150 seizures during the past three days and although the attacks have been quite severe and always general, the muscular

rigidity is most pronounced on the left side which is now decidedly paretic in upper and lower extremity; paralysis is most marked in left leg. Left knee-jerk is markedly exaggerated and ankle clonus is also present; gait spastic-ataxic on left side. The weakness of left side

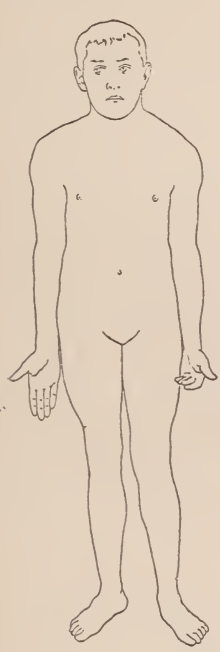


FIG. 13.

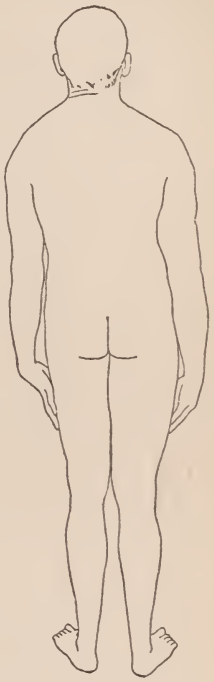


FIG. 14.

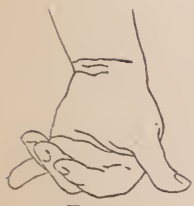


FIG. 15.

includes face and tongue. Patient has had the following number of attacks by years:

1896–97	420
1897–98	1188
1898–99	858

Text-Figure 12 drawn from a photograph taken while patient was in convulsion shows him in one of his *severe general convulsions* in which *consciousness* was *retained*

throughout and following which the customary left side paralysis was manifest for a few minutes. The retention of consciousness in this case in grand mal attacks is of quite frequent occurrence. The convulsions of left side *remained tonic longest*, thus proving the parts ultimately paralyzed were *most* convulsed. The original photograph is shown in Plate I, Fig. 2.

Figures 13 and 14 are from photographs taken in the interparoxysmal state, and show that the muscles of the left side are in slight rigidity and left hand assumes the athetoid position but without mobile spasm. Figure 15 is from a photograph of a hand posed to more clearly present the position of the left hand in Figure 13. The condition shown in these figures has developed slowly after his many exhausted states, and has been permanent for the past six months.* (See Plate I, Figs. 1 to 4).

Case 5.—C. P., Italian, male; æt. 13. Patient came under examination and observation Sept. 10, 1898. Epilepsy began at three years of age. Attacks were grand mal in character from beginning, occurring about twice a week. Dynamometer: right, 42; left, 52. The photograph, Plate II, Fig. 1, shows the patient's excellent physique.

January 7, 1899.—At 10 A. M. patient had a severe convulsion. Another at 10.16; a third at 10.55. After this time until 11.20 patient was more or less in a continuous spasm. At 10.35 the pulse was 139; temperature 100° F. The convulsions began and were most prominent in the left side, the muscles of the arm and forearm being most convulsed and those of the neck, face and leg being least involved.

The spasm was clonic in nature and rhythmic, occurring about seventy times per minute, each convulsive movement consisting of one tonic contraction of the muscles involved. When the movements increased in rapidity and intensity, as they did every three or four minutes, the right side participated in the convulsion to a slight extent.

* March to September, 1899.

At times the clonic spasm was severe enough to elevate the left arm and leg and throw them about for a space of six or seven inches. The face was deeply congested, as was also the whole surface of the body. Profuse general perspiration attended the epileptic phenomena. The temperature was at no time elevated beyond 100° F. For the most part it remained normal. The pulse reached a maximum for the interparoxysmal state of 120 at 11.30. Respiration remained about normal throughout. At 10.35, chloral 20 gr. and bromide 20 gr. were given by enema. At 11.10 a hypodermic of pot. bromide was given in the right breast. After the spasm ceased the patient remained in a comatose state until 11.40 when he became partially conscious and quite restless. At 2.30 P. M. observations in regard to paralysis were made and it was found that the whole left side was paretic, being most marked in the hand. Comparative hand grasps showed dynamometer, right 35, left 15. That the finer muscular adjustments were lost was demonstrated by picking pins from a porcelain dish. Comparative test showed that sensation of the left side was not materially changed. Slight fibrillary tremor was noticed in right hand. Reflexes of left side were all exaggerated. (See Text-Figure 16).

Observation twenty-four hours after the last convulsive movement still showed slight paretic condition in left hand, but was decidedly less than at the first observation.

One week after, the paralytic phenomena had entirely disappeared and no evidence of them remained in reflexes.

October 10, 1899, patient had a severe attack, a typical grand mal seizure of classic epilepsy, which was characteristic of his usual paroxysmal attacks, between the imbricated status periods. After this attack he slept about fifteen minutes. At 9.25 A. M. a peculiar attack occurred as follows: the seizure was entirely confined to the eyeballs, there being no winking of the eyelids. The palpebral fissure was about at normal, but the eyeballs were symmetrically rolled up so that only the whites of the eyeballs presented. They remained in this position

with only slight tremor or vertical nystagmus for about two minutes. Patient was conscious all the time, and tried to correct the malposition of the eyeballs by rubbing his eyes. The convulsion was not painful, but as the patient could not see, he asked to be led to a seat, where he sat down until the eyeballs returned to their normal position. In eight minutes another similar attack occurred.

At 11.05 A. M. the same day, the patient had a severe convulsion, beginning in the following manner: he

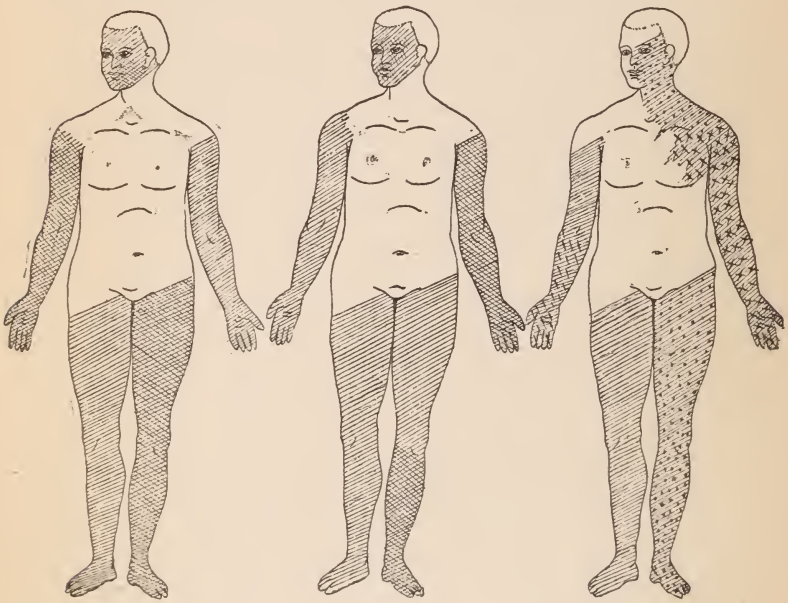


FIG. 16. FIG. 17. FIG. 18.

//=slight convulsion. #=severe convulsion.
::=slight paralysis. ×=severe paralysis.

clapped his hands, and raised the left foot from the floor; he hopped backward on the right foot for four or five feet, and finally fell to the left side bruising the back of the head. The attack was general, consisting of tonic and clonic convulsion; but in ceasing, the clonic convulsion ended on the left side of the mouth, and about the left eye; the left foot and hand were next in order of cessation.

The entire attack lasted two minutes. No paralysis could be detected at this time. (See Text-Figure 17). Consciousness was lost slowly but returned quickly, the patient was at his work again at noon.

At 1.35 P. M. patient was again affected with a severe convulsion. The attack began without the usual premonition or aura. Just before the convulsions became prominent, a marked ring of pallor was noticed about the mouth and on the tip of the nose. The cheeks and neck were deeply flushed. The first convulsions were clonic shutter-like movements of the eyelids of both sides, with an entire facial involvement following rapidly after,—the left side facial movement being most exaggerated; the mouth was drawn spasmodically to the left in quick jerks. The convulsion then extended to right hand and arm, beginning in the arm, extending to the forearm and lastly to the hand. The left leg next became involved. All muscular spasm up to this time was clonic in nature, except in the right arm, forearm and hand; the right arm was in tonic convulsion and continued in this form of spasm, while the other parts were in clonic movements. Finally, the left arm passed into clonic spasm, and then the right leg rapidly followed in the same form of convulsion. The convulsion had now spread over the entire body, zigzagging across in its successive involvement of the different members of the body in the following manner: Both eyes, most in left; left face; right arm, left leg, left arm, right leg. Part was in tonic spasm and part in clonic spasm at the same time. The left leg and right arm participated most in convulsions. The right hand was clenched while the left hand, participating but slightly in convulsion, remained open. Text-Figure 18 shows the predominance of the right hand and left leg in convulsions. Reliable tests for paralysis of these parts could not be made at this time. In a few seconds the general convulsion ceased, and the seizure continued to alternate first on one side, then on the other, in sharp clonic spasms every few seconds; thirty or forty occurring on one side in two or

three minutes, before changing over to the other side. In about ten minutes the clonic convulsive phenomena gradually lessened in severity, then the whole character quickly changed to general tonic, tetanoid or statuesque seizures.* Soon the general tetanoid seizures changed to unilateral tetanoid, and then these spasms alternated from one side of the body to the other, for a short time, as did the previous clonic spasms, then, the upper extremities alternated with the lower extremities in periods of tonic rigidity.

In general seizures the right arm and left leg were noticed to be synchronously involved. The convulsions in general throughout, were accompanied by the usual symptoms of a classic epileptic attack, when not specified to the contrary.

A raised bright erythematous rash was plainly visible, about the sides or margin of the face, and on the neck; it was not hemorrhagic in origin, as is commonly seen in some cases of epilepsy. The rash did not appear about the mouth or on the tip of the nose, which constantly presented the bloodless appearance characteristic of frost bite.

Observations from 3.00 P. M. until death at 8.30 P. M. are as follows: At 3.00 P. M. the convulsions had been in more or less constant display for one hour and twenty-five minutes, and patient had long since entered the typical convulsive stage of status. The temperature rapidly rose; at 5.00 P. M. it was 106.2° F., pulse 160, and respiration 68. At 3.30 P. M. patient was in general convulsions again, and photographs were taken which show many interesting phenomena. On Plate II, Fig. 2, shows the convulsion predominating in the right arm and left leg and Fig. 3 taken shortly after Fig. 2, shows contractures of these parts in the so-called post-epileptic paralytic equivalent which lasted for a few seconds only at this time. In the short period following a few of the general seizures, the right arm was found to fall as a "paralyzed arm" when

* For description of this peculiar form of convulsion, see Clark, "Tetanoid Seizures in Epilepsy," *American Journal of Insanity*, April, 1899.

raised, in marked contrast to the absence of such behavior in the left; at infrequent intervals the left leg showed the same phenomenon. This phenomenon was always most marked in the foot, in marked contrast to the more frequent paralysis which was most prominent in the right arm. Many varying grades of these paralytic manifestations were noticed in the right arm in the semi-conscious states, such as tremors, slowness of movements and incoordinate acts.

At 4.00 P. M. patient passed into deep coma, the "white circle" about mouth became more noticeable as the general cyanosis increased. At 4.20 P. M., at intervals of every two minutes, carpo-pedal contractures occurred, and lasted for two hours, when true tetanoid or statuesque seizures returned again. The arms were extended, all muscles stood out plainly, the hands were clenched, and rotated inward, then outward. The eyes during the convulsions were wide open and staring; between attacks they were in slow rythmic lateral nystagmus; the lower limbs were extended and rigid, while the feet were in the infantile eclamptic posture. These attacks occurred every few seconds, during a period of half a minute; and were rapidly asphyxiating the patient; they suddenly ceased at 7.00 P. M. and there was a return to complete left side spasmodic flexure of the upper and lower extremities. This convulsive state continued for half an hour, flexions occurring every few seconds. Between convulsions the right arm was flaccid, but the left foot below knee continued rigid in the position of pes equinus varus, (Plate II, Fig. 3) until death at 8.30 P. M. There was involuntary defecation and micturition in the statuesque or tetanoid attacks. Patient died from asphyxia in the beginning of a general tetanoid convulsion.

Some fifteen minutes before death, advanced cardiac and respiratory paralysis were manifest; respiration was largely thoracic and of the Cheynne-Stokes type. Hypodermics of bromide, enemata of chloral and bromide, and chloroform anæsthesia were all given without material benefit. No autopsy could be obtained.

I wish to call attention to the varied forms of convulsions and the rapid desultory fire, as it were, of the different cortical centres. It should be noted, that although this status period differed from the left side clonic spasm of the left arm and face before described in this case, yet here we meet with general convulsions, with left leg and right arm, the parts ultimately paralyzed, predominating, which was manifest in the early contracture, in the later flaccidity, and finally in persistent left leg rigidity. A casual observer seeing but one or two convulsions, would have pronounced this case anomalous, (*i. e.*), right arm and left leg paralysis with general convulsions. But we see the case in the true light after careful study.

Again, the seizure at 9.25 A. M. on October 10th, was interesting, so also was the pallor about mouth and on tip of nose, and the erythema.

Finally, the patient passed from comparative health to death, in seven hours, an unusually rapid termination; dying of asphyxia produced by the epileptic paroxysm, a rare form of death for an epileptic.

Exhaustion-paralysis may be an infrequent phenomenon, appearing only once or twice in a patient's epileptic career. The following case showed but one Jacksonian, or better, partial epilepsy, in a long period (ten years) of grand mal seizures.

CASE 6.—F. T. R., æt. 19. Clonic spasm began in the right facial muscles extending to the right arm. It continued for three minutes without loss of consciousness; paralysis and loss of sensation continued for fifteen minutes after the convulsion in right arm and especially prominent in the hand as shown in Text-Figure 19; improvement in both was simultaneous. Right wrist-jerk and left knee-jerk were exaggerated immediately after attack. Ten

minutes after seizure, the right knee-jerk and left wrist-jerk were exaggerated; one hour after attack all reflexes were obtained equally and subtended the normal inter-paroxysmal arc, but the responses were markedly slowed for twenty-four hours. The face did not participate in the paralysis.

In the following case the paralysis was not sudden in its onset nor did it follow the first epileptic attack, but

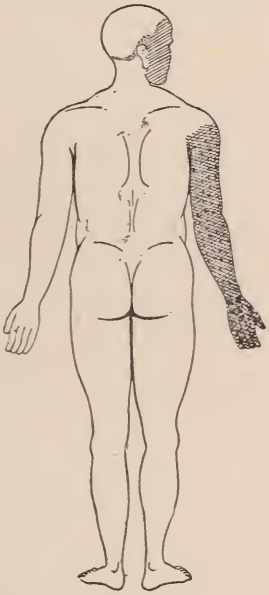


FIG. 19.

//=slight convulsion. #=severe convulsion.
::=slight paralysis. ×=severe paralysis.

came on gradually beginning with the second or third day of the series and the patient gradually lost successively, less complex muscular movements until true complete paralysis obtained. It was very easy to recognize and diagnose the condition after partial recovery had begun. The case presented the clinical aspects of status epilepticus.

Case 7.—F. R., male, aged 53; iron-worker by occupation; age at onset of epilepsy, 50 years. The cause was ascribed to grippe, but the patient had undoubtedly used alcohol to great excess. No neurotic history was obtainable. His seizures were not preceded by any aura. The reflexes, both superficial and deep, were found to be nor-

Fig. 20.

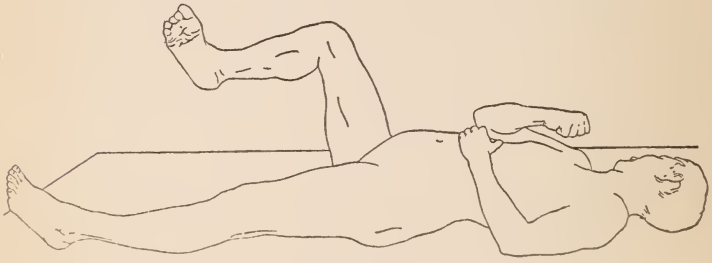


Fig. 21.

Fig. 22.

mal at the time of admission, March 25, 1896. There was a slight muscular tremor in hands and lips.

On April 6th he began to have, on an average, twelve seizures in twenty-four hours. After the first two days these attacks were followed by a partial hemiplegia of the right side; after the first six days he was able to move

his fingers only. The epileptic seizures were always of a general convulsive character. For the first few days, and whenever these seizures would cease for two or three hours, he could perform some of the very simple movements of the arm and leg of the affected side. Fig. 20,* pose of F. R., shows patient with seizure involving left side first and beginning to involve right side of head. Fig. 21 of the same convulsion shows the seizure finally ending on right side in the extreme tonic convulsion, proving the right side most involved in point of intensity. Fig. 22 shows the same patient some days later in convulsion with the right side put entirely out of action, as it were, because of apparently complete exhaustion of that side remaining from previous attacks like those shown in Figs. 20 and 21. The left side alone remains capable of carrying on the convulsion phenomenon in subsequent seizures. The deep reflexes of the right side were very much exaggerated; all through his series of attacks ankle clonus was a prominent symptom. Plantar reflex was always obtainable and was markedly exaggerated toward the end of the series.

During the nineteen days following the first attack, he had 249 distinct and separate convulsive seizures. The table on following page gives a more accurate conception of the order in which the seizures occurred.

April 21st he began to have fewer attacks daily, and on the 25th he had only one; for the next eight days he had but one attack a day. At the time he began to have but three attacks a day, he slowly picked up some of the less complex and comparatively simple muscular movements. Throughout the entire period, the superficial and deep reflexes remained exaggerated and much more noticeable on the affected side. A small corneal ulcer made its appearance on the 24th and a bedsore began on the right buttock the next day. Both were healed very quickly by local application During this time patient lost twenty

* The negatives taken from the actual convulsion were too faint to allow of reproduction, but were used as guides in these poses (of figs. 20, 21 and 22) which are particularly accurate.

RECORD OF SEIZURES OF CASE 7 (F. R.) FOR 19 DAYS.

Number of Seizures in each Attack.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	Total No. of Attacks.
April 6	..	..	..	..	..	..	..	1	..	1	..	..	..	..	1	1	..	..	..	..	..	..	..	..	4
7	1	..	..	1	.	..	1	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	4
8	3	2	2	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1	9*
9	1	3	1	2	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	9
10	..	1	3	..	3	1	..	..	..	1	..	..	1	..	..	1	..	..	..	..	..	..	1	2	14
11	1	2	2	2	..	1	..	2	..	..	..	1	1	2	..	..	1	..	..	1	..	..	1	..	17
12	..	..	1	..	..	..	1	1	1	1	..	1	1	..	3	1	..	1	..	..	..	1	..	..	13
13	1	3	..	1	..	1	1	1	3	1	1	..	2	1	1	1	1	..	..	1	..	..	2	1	23†
14	1	2	2	2	1	2	..	2	1	1	..	..	1	..	..	..	1	..	..	1	1	1	2	2	23
15	1	..	..	..	..	..	1	1	2	..	1	2	1	1	..	..	1	..	..	1	..	..	..	..	15
16	..	..	1	..	1	3	1	1	1	..	1	..	1	..	1	..	1	1	..	1	..	..	3	..	17
17	2	1	2	1	..	..	1	1	.	1	..	..	..	..	1	..	1	1	..	..	..	1	1	..	14
18	1	..	2	..	..	1	1	1	1	1	..	..	..	..	1	1	..	..	..	..	..	..	..	3	13
19	1	1	1	1	1	..	1	..	..	1	..	..	3	..	..	1	3	1	1	..	..	..	1	1	18
20	1	..	2	..	1	..	1	..	1	..	..	..	..	1	..	1	..	..	..	..	..	..	1	..	9
21	..	1	..	..	..	1	..	..	..	1	1	..	1	1	..	..	..	..	..	..	..	..	..	2	8
22	1	..	1	1	..	3	4	..	..	..	..	..	1	..	1	3	1	2	..	1	..	..	1	..	20
23	..	2	..	1	1	1	..	..	..	..	1	..	..	1	..	1	1	1	..	..	..	..	..	2	12
24	1	1	1	..	..	..	1	..	2	..	..	1	..	.	..	..	..	..	..	..	..	..	..	..	7
Total Number of Seizures.	16	19	21	12	8	14	14	11	13	9	5	5	13	7	10	12	11	7	1	6	1	3	13	18	249

* Followed by partial right hemiplegia.

† Followed by complete right hemiplegia.

pounds in weight. From April 24th to May 15th he made uninterrupted progress toward recovery of almost all of the muscular movements, but there still remains some slight paralysis of the more complex muscular movements. The deep reflexes of the left side at first were less than the right and below a normal reflex. This condition persisted until the stated improvement began; then they became exaggerated equally with the right side.

An examination of the patient made in the latter part of May, 1896, showed that the right side was still a little weaker than the left and all active muscular movements were a little less co-ordinate as compared with those of the left. Muscular movements are uncertain and awkward, showing that certain complex and delicate functions of the muscles have been permanently lost. All superficial reflexes—epigastric, axillary, cremasteric, plantar, scapular are exaggerated. The deep reflexes of both sides are considerably exaggerated, the right much more than the left, and ankle clonus was present on the right side. There is present in the hands, lips and tongue a slight, rhythmical, fibrillary tremor. The right foot drags, and swaying to the point of falling is still present. Patient experiences no pain either in passive or active muscular movements, but is occasionally troubled with what he terms rheumatism in the shoulder muscles. On measurement no muscular wasting was found, although the muscles of the affected side were flaccid and soft to the grasp when compared with the left.

Patient's condition six months after his paralysis is as follows: Knee-jerks of both sides are exaggerated, the right more than the left; ankle clonus is still obtainable in both sides, although is much more marked on the right; both wrist-jerks are markedly exaggerated, but the right is much greater than the left. The right pupil is inco-ordinate in its adaptation to light and the patient is inco-ordinate in his attempts to perform many of the more complex muscular acts involving muscles of the affected side. He states that the reason why he cannot perform

these acts as well as he could at one time is, that he is now "very awkward and clumsy."

An examination made in April, 1898, (two years after his attack), showed complete recovery of all the paralyses. Several attacks occurring in series since, attended by temporary paralysis, proves exhaustive nature of the resulting paralyses. Patient is at present (October 1, 1899) doing a full day's work at his trade—blacksmithing—having occasional isolated epileptic fits.

The following case illustrates how a paralysis, induced and of a lasting nature though not noticeable during vigorous health, may become very prominent and easily recognized whenever the patient becomes weak and feeble physically, as this case was at the time of his admission to the Colony; and again, how these symptoms of cerebral lesion disappear in the main when the general physical condition improves.

Case 8.—V. S., age 29 years; epilepsy began at the age of nine; a well marked history of family degeneracy was obtainable. Patient's father died of general paresis. At the time of his admission the inequality of patient's pupils was quite marked, the right being the larger and responding less readily to the accommodation test than the left, although a decided reaction takes place in both. His gait was spastic-ataxic. Some rigidity and spasm of muscles was found at all examinations, although most marked in right leg and foot. The disorder of gait was doubly noticeable when he attempted to walk with the eyes closed. All active motions of upper and lower extremities were awkward, uncertain and inco-ordinate. Passive motion of the extremities was materially interfered with because of the muscular spasm; there was greater spasm in the right arm and hand than in the left. Tactile sensibility, pain, thermic sense, and localization of sensation were normal,

although slight retardation of sensation was noticeable along the outer side of the right leg, especially prominent before sensation had become schooled by examination. Cutaneous reflexes were normal. Myotatic irritability was greatly exaggerated on both sides, but was most marked on the right. All the other so-called reflexes of elbow and wrist were exaggerated and most prominent on the right side. His speech was monotonous, scanning, and of a nasal tone.

The most noticeable features of his entire nervous examination at the time of admission were the spastic-ataxic gait, inco-ordination in upper and lower extremities, exaggerated knee, wrist and elbow-jerks and foot clonus, all these symptoms being largely confined to the right side.

These symptoms were so prominent that several neurologists pronounced the condition a lesion in the cerebral cortex; the writer agreed with this conclusion until it was found that the patient had previously suffered from two attacks of post-convulsive paralysis. Prior to admission, he had averaged one hundred seizures a month for five months, and during the first month after his admission he had one hundred eighty-three attacks and was hardly able to walk without support. Since admission he has gained twelve pounds in weight and is working daily in one of the shops at the Colony. He has had no seizures for five months. Examination at the present time shows pupils normal; knee, wrist and elbow-jerks on the right side are still exaggerated and foot clonus is very slight. Inco-ordination has disappeared and little or no disturbance of gait is noticeable. Whenever for a few days he has suffered from any severe physical exhaustion —from epileptic attacks, while they were present—the symptoms of his nervous disorder of exhaustion-paralysis become quite prominent.

The patient has had no epileptic attacks for over three years and has been discharged cured. He is now earning a livelihood as a printer, a trade which he learned while a patient in the Colony.

Permission has been obtained for an autopsy in this case, even if dying from any condition other than epilepsy. Many cases of cured epilepsy of this character, if carefully studied pathologically, would be highly instructive. Comparative study of the mental and physical regeneration with that of a possibly visible brain repair is to be considered most essential.

The following cases (9 to 13 inclusive) illustrate exhaustion-paralysis in infantile cerebral palsy:

Case 9.—G. E., æt. 27. Epilepsy began at the age of five from an infantile cerebral palsy which left the right side partially paralytic. All reflexes are exaggerated on right side and ankle clonus is obtainable. Epileptic attacks generally occurred in series. Relative hand grasp in interparoxysmal state: left, 65; right, 45. Atrophy and contracture on right side is most manifest in lower extremity.

Twenty observations were made upon this case. Tonic convulsions begin in the paralyzed side (quadriceps muscle) and quickly become general, ending by clonic spasm in the right or paralyzed side. During the convulsive stage of all attacks, the face is drawn to the left; consciousness is lost; there is stertor for a few seconds. Attacks last for 1½ minutes; immediately after attacks right reflexes are lost, to be regained slowly at the end of eight or twelve minutes, and to be at last exaggerated, as is usual in infantile palsy cases. Complete paralysis is manifest for fifteen minutes, in excess of the usual partial, interparoxysmal palsy, lasting for ten hours, which is noticeable in the marked effort to maintain equilibrium and to use the right leg in walking. Face is not paralyzed

immediately after seizure. Relative hand grasp: left, 60; right, 25.

Text-Figure 23 presents the areas of the convulsive and paralytic phenomena, while Text-Figure 24, drawn from photograph of patient shows the right side in a hemiplegic state, most manifest in right arm, and least of all, in leg where convulsion first begins.

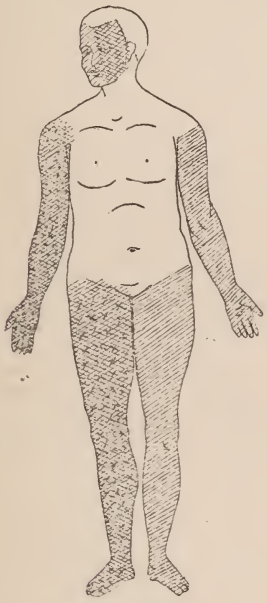


FIG. 23.

//=slight convulsion.
::=slight paralysis.

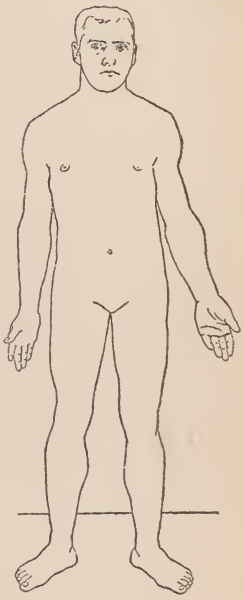


FIG. 24.

#=severe convulsion.
×=severe paralysis.

CASE 10.—J. L., æt. 16. Epilepsy began at two years of age, ushered in by a cerebral palsy leaving right side completely paralytic for several months. The palsy is hardly noticeable at present. The palsy was caused by a severe attack of diphtheria.

Serial attacks of general convulsions most marked on right side, eight or ten in twenty-four hours, leave the right side completely hemiplegic from exhaustion for several hours. In the course of five or six days after

serial attacks the hemiplegic side regains the power normal to it in the interparoxysmal state.

Case 11.—T. D., æt. 13. He has had epilepsy of grand mal type since infancy and on the average three attacks per week. His epilepsy probably dates from an infantile cerebral palsy of the left side of the body at the age of three. There are internal strabismus of the left eye, and multiple hereditary and acquired stigmata. At periods he has been subject to "laughing attacks" of epilepsy. While his attacks are general, they begin and predominate on the left or paralyzed side. Generally, after a series of forty or fifty attacks in forty-eight hours, paralysis is complete on left side for twelve to fourteen hours. Several days' freedom from epileptic attacks is necessary for complete restoration to interparoxysmal state of average motility of the left hemiplegic side. The paralytic condition is hardly noticeable in the interparoxysmal state.

Case 12.—G. C., æt. 12. Epilepsy dates from infantile cerebral palsy at sixteen months of age which rendered the left side hemiplegic. Patient has always had seizures predominating on the left side. When the seizures are many, paralysis returns to its complete form, to be recovered from very slowly through a period of several days.

The following is a description of the attacks in one of the series (November 28, 1898) in which the paralysis was most severe: Epigastric aura and epileptic cry. Tonic convulsions began in upper extremities first; rigidity was most marked in the left arm. The fingers of the right hand took the "convulsive position;" forearm was flexed on arm; left hand was partially closed; left forearm and arm were flexed as in the right, but engaged in a more prolonged tonic spasm. The head was turned to the right. The tonic spasm then involved the lower extremities in symmetrical manner from the proximal to the distal end. Extremities were flexed at all joints, but while the right leg passed into the clonic stage the left leg was slowly extended and remained straight and rigid until the right

side clonic movement ceased. The convulsion ceased simultaneously bilaterally. Tonic stage, 25 seconds (for right side, 21 seconds). Clonic stage for right side was 4 seconds. Stertor lasted for 30 seconds. Consciousness, which was lost at epileptic cry before convulsion, returned in four minutes, after which he walked about with difficulty. This attack was the third in a series of twenty-three seizures occurring in twenty-four hours, after which complete left paralysis obtained for several hours in left side as shown in Text-Figure 25.

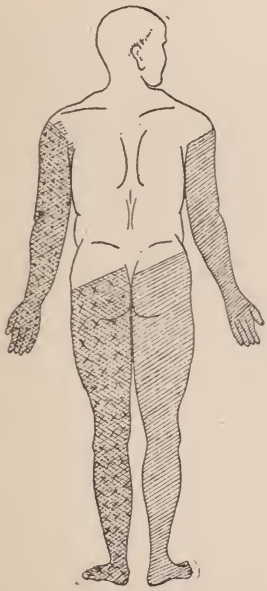


FIG. 25.

//=slight convulsion.
::=slight paralysis.

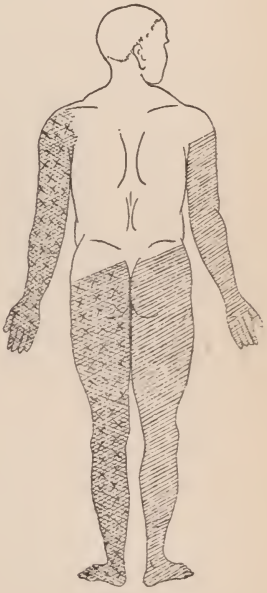


FIG. 26.

#=severe convulsion.
×=severe paralysis.

CASE 13.—S. A., female, æt. 23. Epilepsy dates from infantile cerebral palsy at six. Left side is hemiplegic. A permanent weakness remained most marked in the left arm Speech is ataxic and appears to have suffered in early palsy. Muscles of paralyzed side are flaccid; considerable wasting is noticeable. Convulsions begin in the thumb of left

hand and extend to muscles of the right side, and finally to left leg. The manner of the termination of convulsions is always in inverse order to its onset, thus convulsing the left side most, although patient usually remains in prolonged stupor after an attack. The paralysis of left side is most marked after the seizure and is gradually recovered in the course of the day. Patient died in status from exhaustion. Before death paralysis of the left side was absolute. Sloughing bedsores formed on the left side, quite analogous to those seen in the last stages of general paresis. (See Text-Figure 26).

Case 14.—A. S., male, æt. 28 years. Admitted to the Colony, May 22, 1896. Epilepsy began at twelve years of age. Supposed cause was ascribed to cranial traumatism.

Patient's attacks have always been the grand mal type, characterized by a leisurely and well marked order of invasion. It was thought that he was suffering from Jacksonian epilepsy, consequently he was trephined over the right motor region. Notwithstanding this operation, patient continued to have the seizures as before. With this exception that, where the attacks formerly were all grand mal, now, a few are Jacksonian, so far as localization is concerned.

The following description of a partial attack, is one out of three or four similar attacks which he has had while under observation at the Colony. He is undoubtedly a case of idiopathic epilepsy.

Patient had an unusual attack at 3.45 P. M., March 24, 1899. The seizure was partial. There were no premonitory symptoms and no aura. Patient lost consciousness at the onset.

The order of muscular invasion was in the muscles of the chest, the left arm, and then the forearm.

The thumb flexed in the palm and the fingers took the common convulsive position of epilepsy. The convulsion ceased in the same order as the onset. The left eye was closed and the eye-ball was turned to the left and slightly

upward. There was a very rapid clonic movement in the facial muscles about left eye. The lower jaw was drawn toward the right and the head to the left. The pupils were contracted throughout and for one minute after the seizure. (See Text-Figure 27).

Patient had sensory and motor paralysis of the left hand. The sensation returned as though the hand had recovered "from being asleep." Return of sensation was complete

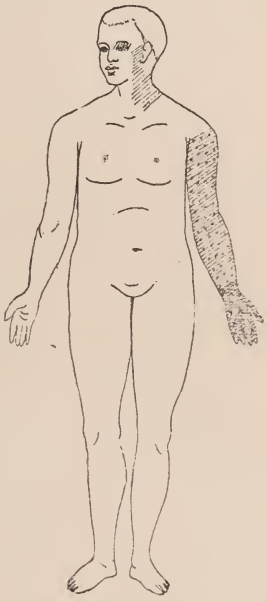


FIG. 27.

//=slight convulsion. #=severe convulsion.
::=slight paralysis. ×=severe paralysis.

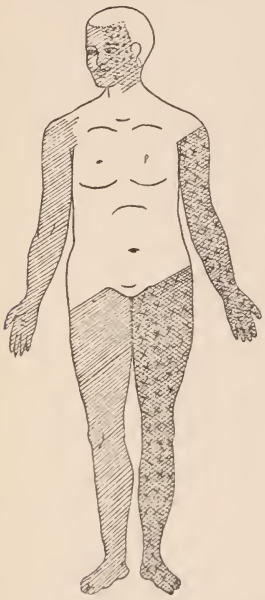
in two minutes, while a slight paresis remained in the left hand for six minutes following the attack. There was no coma nor stertor following the muscular convulsion. Left wrist and knee-jerk were exaggerated one minute after the attack, the exaggeration being most prominent in the wrist.

The grip in the right hand was twice as great as in the left five minutes after the attack. Although the sensation

and motion returned in six or seven minutes, the left hand remained cold and cyanosed for fifteen minutes.

The following case is interesting, as exhaustion-paralysis occurred in the left side (suffering from infantile paralysis from the age of three) and, at the same time, exhaustion-paralysis pure and simple occurred on the right side (not previously incapacitated from any brain lesion). An excellent opportunity was presented to compare the exhaustion of the two sides and their recovery rate.

Case 15.—M. E. H., female, æt. 12. Epilepsy began



Fig, 28.

//=slight convulsion.
::=slight paralysis.

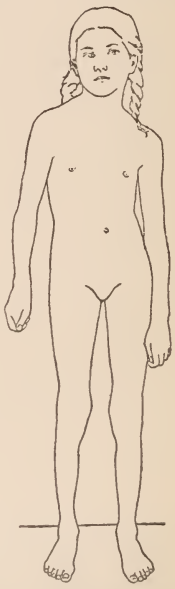


Fig. 29.

#=severe convulsion.
×=severe paralysis.

when patient was 4½ years old. Neurotic family history. Seizures dated from infantile cerebral palsy with left hemiplegia. During the first two years of epilepsy, the convulsions were confined to the left side, but since then they have been general. For the past two years, patient has had

occasional petit mal attacks. There was a loss of speech with left side palsy. Patient recovered sufficient power in hemiplegic side so that she was able to walk about to some extent after five months from the onset of palsy. Of late several attacks have occurred daily. At various periods during the past two years patient has had series of eight or ten convulsions in forty-eight hours, the left side being chiefly involved. An after-paralysis nearly complete has lasted from one to two days. Atrophy is

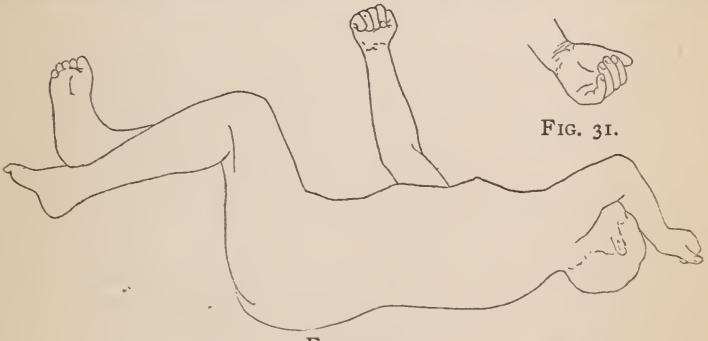


FIG. 31.

FIG. 30.

most marked distally; there is but little contracture. Comparative lengths of extremities of both sides are equal. All reflexes of left side are exaggerated.

In July, 1898, typical exhaustion-paralysis supervened upon left (palsied) side, leaving that side completely hemiplegic for weeks. Right side remained unaffected. Seizures convulsed left side most, beginning and ending on the left side. (See Text-Figure 28).

The exhaustive phenomena of right side and especially of right arm, was first manifest in the series of attacks from September 28th to October 8th, 1898. The temperature September 30th reached 106° F., pulse 168, respiration 60; two hundred sixty-three seizures occurred that day mainly localized to right side and predominating in right arm. This is admirably shown in Text-Figure 30 drawn from photograph reproduced on Plate IV, Fig. 2. The left arm remained free from convulsion. Text-Figure

31 is from a pose showing more clearly the passive position of the left hand of Fig. 29. Various modifications of exhaustion phenomena manifested themselves during this period.

October 8, 1898.—Fingers of the right hand assume a state of athetoid spasm (Strümpell); index finger is extended; thumb flexed to the side of metacarpal phalangeal joint and over third and little fingers in semi-flexion at second joint. Third and little fingers are flexed only at the knuckle; other joints of fingers are nearly straight. Continuous slight fibrillary tremor is found in all fingers, especially the third and little fingers. Earnest voluntary effort enables the fingers to be partially extended at the knuckles, but such effort is attended by some pain and performed very slowly. Painful contracture which complicates palsy is called by some the post-epileptic paralytic equivalent. Text-Figure 29 is drawn from patient's photograph taken Oct. 12th, 1898, and most excellently presents the old hemiplegia of the left side and the exhausted state, especially seen in right hand; the contracture weakness and inco-ordination were still marked at the time of taking this photograph, which is reproduced on Plate IV, Fig. 1.

November 27, 1898.—Relative hand grasp after a seizure which occurred at twelve o'clock noon:

12.20 p. m., R.....	12,	L.....	15
12.35 p. m., R.....	14,	L.....	15
6.04 p. m , R.....	18,	L.....	17

On December the 2d patient had severe attacks at 8.32 A. M., 9.20, 9.27, 10.00, 10.10. Had carpo-pedal contractures lasting for two or three seconds and reappearing every seven or eight seconds. Thirty-seven of these occurred in seven minutes immediately after last seizure. Temperature, 99½; pulse, 112; respiration, 24. No paralysis was present after this series.

December 7th.—Severe attack at 12.22 P. M. followed by carpo-pedal contractures lasting for two or three seconds and reappearing every seven or eight seconds. This

condition remained general for sixteen minutes but was confined to right side for twenty minutes, and was more marked in the right arm and hand. Patient was very irritable and noisy at times during the day. Contracture, rigidity, and tremor of right hand after seizures.

December 14th.—Paralysis continues most marked in right upper extremity. The right hand at rest frequently assumes Strümpell's typical athetoid position. The movement of the fingers are mostly those of mobile spasm; no tremor is noticed; there is much less contracture than formerly. Upon strong volitional effort, considerable power is manifest in the hand. Right shoulder and arm muscles still appear weak. The extremity as a whole is lifted very slowly and with great effort. Right hand is cold and the circulation is very poor.

January 2, 1899.—Patient had a slight attack at 9.30 A. M., and a severe convulsion at 4.24 P. M., followed by fifteen carpo-pedal contractures occurring about once in a minute and continuing to grow less severe toward the last. The right arm and leg kept up a continuous trembling motion for one hour. The left leg was affected in the same manner for a few seconds.

January 4th.—Slight attacks at 11.20 A. M., 12.20. P. M. and 6.15. Those at 12.20 and 6.15 were followed by carpo-pedal contractures of the right side occurring every twenty seconds and lasting for fifteen minutes, followed by complete right foot and hand paralysis and contracture for two hours. See Text-Figure 32.

January 7th.—Slight convulsion at 3.00 P. M. followed by carpo-pedal contractures, affecting the left side most; continued for ten minutes. Motility of the right side was unchanged; the left side was markedly weakened for half a day. See Text-Figure 33.

January 8th.—Severe convulsion at 8.45 A. M. and 3.25 P. M. Both convulsions were similar in character, affecting the left side most, although apparently general. Patient's head was drawn downward and to the left side. Thumb and fingers were flexed on palm, wrist flexed and arms flexed at elbow, arms drawn upward with hands in the

vicinity of the face. Toes were flexed and feet inclined forward from ankles, legs were flexed on trunk. Patient turned on left side and very soon on face, the body being twisted from the waist upward. She was forcibly turned on her back, the eyes were closed, and face very deeply congested; muscles then relaxed, limbs were partially straightened and a few clonic spasms followed. Tonic stage lasted one minute; clonic, only a few seconds; stertor about 1½ minutes. Soon after the tonic stage the patient uttered a peculiar cry. The last convulsion was followed

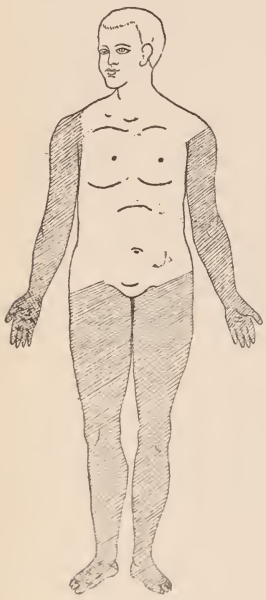


FIG. 32.

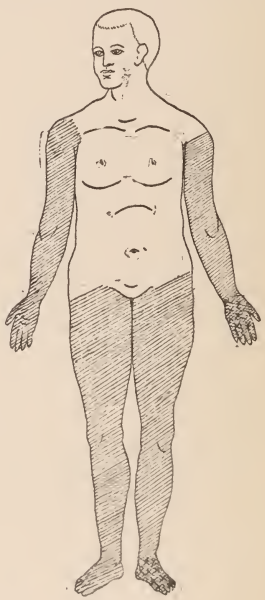


FIG. 33.

//=slight convulsion. ,',',=tremor. ×=severe paralysis.

by carpo-pedal contractures, beginning immediately after convulsion and continuing for thirteen minutes, gradually diminishing in severity towards the last. During the first six minutes the left arm and right leg were most affected; afterward the right side alone was involved. The toes of the right foot in hyperextension, foot inclined inward from ankle, leg flexed at knee—(this position was taken

suddenly, the leg being immediately straightened when contracture subsided). The right hand was semi-flexed and the wrist completely flexed, the arm remaining straight.

Slight convulsion at 3.00 P. M. followed by carpo-pedal contractures affecting the left side most; continued for ten minutes. Slight weakening of the left side; the right side remained unchanged.

The degree of the paralysis of the left side is dependent on the severity and frequency of the attacks. Cessation of attacks for a few days allows the side to return to the normal interparoxysmal paretic state. Although in severe series the convulsions are confined mostly to the left side at first, they change to the right side almost entirely. They apparently do not do this until there is complete paralysis of the left side. Exhaustion-paralysis then rests upon an organic paralysis, producing complete paralysis.

The cerebral centre of the right side being left alone to carry on the convulsive state is soon exhausted and becomes more or less paralytic, but will recover again more or less completely if allowed to rest for any marked period.

At present, Feb. 10, 1899, patient has two kinds of paralysis which give us excellent opportunities for study. Left side has flaccid, soft and atrophied muscles, giving a spastic gait; right side is in slight rigidity and contracture, giving ataxic gait. This condition shows constant improvement. Patient is practically in the same condition as that shown in Figure 29 although having much more strength and able to be about the hospital ward without assistance as was formerly necessary when this photograph was taken (in October, 1898). Yesterday, after one general and two slight attacks, (the first most severe on the left side; the two last slight on right side most), she could not move the right hand, but to-day is able to write a letter.

The fact that after the convulsions of the left side have exhausted that side it may no longer participate in convulsions, is in full accord with physiological experimentation on cats and monkeys, and finds a clinical analogy in some infantile cerebral palsy cases in which the cerebral lesion is so destructive that the motor centre is not able to take the first prompting to discharge, and the convulsion begins in the opposite side.

Exhaustion, in an easily recognized form, follows so very frequently in infantile cerebral palsy cases (the author has seen many more or less marked cases) that we are surprised that exhaustion phenomena have not received more attention. At first the relative diffusion theory of Jackson seems especially applicable here, but it is far from being analogous. The theory presupposes the localized convulsion to be produced by external means, but we have here an organic lesion producing the phenomena because of its previously weakened state. We see no reason why this case and those of Dutil's showing rigidity and contracture should not rank with that class of organic hemiplegias attended by contracture. It is not necessary to call the contracture an equivalent of temporary exhaustion.

This case has frequently developed a rash closely resembling angio-neurotic œdema; at other times similar to giant urticaria. A list of the dates upon which the rash appeared and the length of time it lasted is given below. In addition to the notes on the appearance and disappearance of the rash, the seizure immediately preceding and the one following it are noted. We are quite certain that the condition has attended the epileptic phenomena, but data do not prove it to be always a forerunner or a sequela.

Last Attack Before Eruption.	Eruption Appeared.	Eruption Disappeared.	First Attack After Disappearance of Eruption.
Aug. 24.	Aug. 26, 1898. On lower limbs at 2 P. M.	Aug. 26, 1898. At 6 P. M.	Aug. 26, 1898. Severe.
Oct. 17, 1898. In early morning.	Oct. 17, 1898. At 2 P. M.	Oct. 18, 1898. 1 P. M.	Oct. 18, 1898. Very severe.
Oct. 18, 1898. Slight.	Oct. 19, 1898. In the A. M. on trunk limbs	Oct. 20, 1898. 1 A. M.	Nov. 6, 1898. Slight, at night.
Nov. 18, 1899.	Nov. 19, 1899. 9 P. M. General Uticaria on trunk, limbs and face; Temp. 96.6; pulse 96; resp. 20.	Nov. 20, 1899. At midnight. Temp. 99; pulse 96; resp. 20. leaving œdema of face, waxy, with slight red margin. 6.30 P. M., temp. 101.6; pulse 112; resp. 32.	Nov. 24, 1898.
Nov. 28. Twelve slight attacks	Nov. 28. 2 P. M. On trunk and limbs.	Nov. 30.	Dec. 1, 1898. Severe, daytime.
Dec. 2, 1898. Five severe attacks.	Dec. 2, 1898.		Dec. 2. Severe attacks.
Dec. 3, 1898. One slight.	Dec. 3. 2 P. M. Few small spots on abdomen.	Dec. 3. At night.	Dec. 3, 1898. At night, four severe.
Dec. 3. Four severe attacks. (Reported above).	Dec. 4. 2 A. M.	Dec. 4. 4 P. M.	Dec. 4. Three severe attacks after disappearance of eruption.
Dec. 9. Two slight attacks.	Dec. 9. 2 P. M. Trunk and limbs.	Dec. 9. During the night.	Dec. 11. At night, severe.
Jan. 8, 1899. Two severe attacks early morning.	Jan. 8, morning. Face, trunk and hands.	Jan. 8. During the night.	Jan. 16. Severe.

February 27, 1899, patient had six right-sided convulsions. During the night of the same day she had 95 of a similar character. On the 28th she had 132 which involved the right side in greater part. The left arm and left leg occasionally moved about in a semi-purposeful manner but there was no muscular rigidity, no clonic or

tonic spasm of left side in this series. March 1st patient was conscious but could not speak, although she made persistent and earnest effort to do so. The entire right side was more or less paralyzed, motion returning in right leg first. (See Text-Figure 34). Efforts to grasp with right hand were attended by associated movements in left side but no movement of right hand; later, motion was obtained. Contrary to the observations of other authors there was increased weakness of left side although it did not participate in these convulsions. Return of

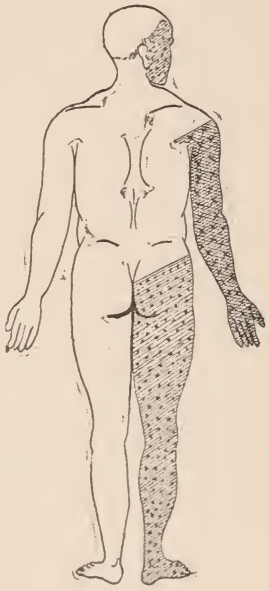


FIG. 34.

//=slight convulsion. #=severe convulsion.
::=slight paralysis. ×=severe paralysis.

motion of right hand was slow and flexion was always preceded by an initial extension in endeavoring to grasp any object. Once flexed, the hand remained in that position for six to eight seconds after efforts to loosen grasp were made (myotonic symptoms). This latter condition speedily improved during the day. The next day (March 2d) patient was able to raise the right upper

extremity from the bed but the effort was attended by violent associated movements of left extremities (upper and lower).

Giant urticaria appeared at 2 P. M. lasting for fifteen minutes.

During this period of status just described, convulsions and paralytic phenomena were completely limited to the right side from beginning to end, a rare condition in infantile hemiplegic epileptics. The contractures were not painful and sensation was not impaired.

March 7th a series of convulsions occurred consisting entirely of convulsions of the following character: Convulsions began in the right side of neck, rotating head slightly to right, eye-balls similarly deflected to right and right facial spasm present; then head, eyes and mouth turned slowly but decidedly to left and finally to right side in complete and forceful rotation. The spasms then began in right arm distally and proximally at once; slight flexion and rigidity occurred, causing the arm finally to be raised as a whole, high above the head. In about five seconds both legs passed into tonic rigidity simultaneously. The left arm remained free throughout the entire series of 439 seizures, except once, to be mentioned later. The convulsive state here is almost identical to that shown in Plate IV, Fig. 2.

At 3.50 P. M. the grasp of the left hand was 25, of the right 14. A severe attack followed the test of the left hand; it was confined mostly to left side and especially to left arm. Five minutes after this attack a slight right-sided seizure took place. Both of these attacks were out of the periods running between attacks and seemed produced in large measure by muscular tests.

Case 16.—A. J. This case is in many respects unique. The exhaustion followed a true convulsive seizure, and paralysis of speech muscles resulted,—a complete motor aphasia for six days. The aphasic condition was noticed a few minutes after recovery from the attack, at which his attempts at speech were chattering and no definite or

recognizable words were uttered. He readily understood all that was said to him, as was evidenced by his ready compliance with one's commands and his answers to questions addressed to him; he wrote his answers on a slate. There was no discoverable paralysis in facial muscles and no loss of power was observed in any part of the body. Patient performed physical work daily and was, to all appearances, usually well and happy. On the sixth day after the seizure on which the aphasia followed, his speech began to return. The first day *l*, *m* and *n* were fairly well spoken, but *p*, *q*, *r*, *s* and *t* were articulated imperfectly and with great difficulty. Of the vowels, *a*, *i* and *o* were plainly articulated but the remainder were imperfectly given. There was considerable puffing of the lips and cheeks at each attempt at pronouncing the few words which were not perfectly uttered; this continued until the latter part of the seventh day. On the afternoon of the eight day, he was able to speak several short sentences in German, his native language, but his utterances were not so distinct and devoid of stammering as formerly. (Patient states that his stammering began after the seizure followed by his first attack of aphasia four years ago). On the tenth day, patient was able to resume normal speech but a hoarseness persisted; this, however, gradually disappeared during the next three days.

There is some question as to whether this attack preceding the aphasia was a true epileptic seizure; a description of it is about as follows: About nine o'clock he was found in a maudlin condition, waving his arms about and appearing like one half intoxicated. His speech was chattering and no recognizable words were uttered. He attempted speech several times during the next few hours but was as equally unable to make himself understood as at the first attempt. Patient states that until eleven o'clock he did not understand anything that was spoken to him. He states that just prior to the loss of consciousness he remembers a patient saying to the nurse "Come quick." He became conscious at about eleven o'clock and made several attempts to speak, but felt as though the

muscles of his throat were paralyzed. His head ached and he felt stupid and languid just the same as he had "after previous epileptic attacks."

About four years previous to his admission to the Colony (March, 1892) he was afflicted with the same degree of loss of speech which lasted for two or three hours and was sequent to three epileptic seizures which came in rapid succession. He had no other paralysis of the body and was apparently healthy aside from the motor aphasia. Since then a slight period of loss of speech has been noticed, lasting only for a few minutes after each attack.

September 12, 1897, 2 P. M., after having had one severe grand mal attack, the patient was left aphasic. This had its dependence entirely upon the motor element, as none of the other forms of aphasia were present. Patient was not able for the first two days to whisper plainly enough so that his speech could be understood. On the fourth and fifth days patient was able to communicate his ideas in a hoarse whisper; on the sixth and seventh days patient was able to partially phonate. All attempts at speech were generally of a hesitant nature; an effort was made almost always on expiration. On the eight day the patient was able to speak several words in German and two or three in English quite distinctly; further than this, although great effort was made, no words were distinctly pronounced. The morning of the ninth day patient was able to speak as in his natural state. No seizure occurred in the interim. The patient presented none of the stigmata of hysteria and seemed to have suffered little mental disturbance other than that of some degree of apprehension for so mysterious a loss of his speech. He was able to be about the Colony and at his work each day during his attack of motor aphasia.

December 3, 1898, 11.30 A. M., patient had a severe seizure. The attack was ushered in by the usual plaintive cry and patient passed through the customary tonic and clonic stages. From stertor the patient passed into a natural sleep which lasted about forty minutes. On

awakening he was found to exhibit considerable lameness on left side and a complete loss of voice. He also complained (by signs) of trouble in head. The lameness made itself apparent for about two hours, and five hours after the attack patient was able to make himself understood in a very faint whisper, which, however, at times became a mere rasping sound. The attempts to articulate were produced with great effort and accompanied by lancinating pain in right lung. This pain also accompanied ordinary breathing. Patient slept well during the night and on awakening in the morning was able to use his voice fairly well, and by 10 A. M. articulation was almost normal, but the patient still complained of pain and weight in chest when speaking and breathing. The pain is now described as if he were being pierced with a blunt pointed instrument. All the other functions appear to have maintained their normal conditions.

March 3, 1899.—Patient became aphasic at 10.30 A. M. He was in his usual good health until 10 A. M., when he complained of feeling ill so he could not work. He states that on sitting down he felt something come up from his stomach to the right temple and as the feeling slowly circled around the head he was dizzy and the scalp felt as though some one was pouring a stream of cold water on the top of the head. This was probably in the beginning an epigastric aura which turned into an attack of sensory discharge purely. Such epileptic seizures are common in certain types. Speech defect was similar in all respects to that noticed September 12, 1897, occurring after a grand mal attack. The condition disappeared almost entirely March 7th, except the hoarseness, when, with premonitory signs of a seizure, he suddenly, as before, lost speech entirely. No convulsion followed. The same symptoms presented themselves as on previous occasions after seizures. At all times patient has been free from stigmata of hysteria.

Cases of partial motor aphasia of two, three and in one case of twenty-four hours' duration have been commented

upon by Hirt, Jackson and Gowers. When the condition has been very transient, lasting for two or three hours only, it has been explained upon the theory of inhibitory action of a higher controlling centre.

In this case of aphasia it is not necessary to invoke inhibitory paralysis, as a seizure actually occurred, thus placing it under exhaustion-paralysis.

Certainly this case in its various manifestations of aphasia proves either the one thing or the other, namely, the condition of loss of speech after convulsive attacks is purely inhibitory, or is of an exhaustive nature, preceded or not by convulsions. I do not hesitate on the side of the exhaustion explanation for the entire condition.

CASE 17.—W. S., male, æt. 20, single. Epilepsy began at ten years of age. Supposed cause scarlatina. No heredity was traceable. His attacks alternate from grand mal to petit mal and occur from two or three a week from five or ten a day. The greater number occur at night. No aura is present. After each attack patient is very active and rarely has any sleep stage. He is well disposed and amiable and performs any work that may be assigned to him. On admission to the Craig Colony the physician's certificate stated that "the patient had had a paralysis of both legs which lasted two weeks, but that there was no paralysis at the present time." (Possibly exhaustion-paralysis).

Upon examination at the Colony it was found that the knee-jerks of both sides were greatly exaggerated and that there was some muscular rigidity of the right side. The right side also showed a slightly greater response in the tendon reflexes. No foot clonus was obtainable. Superficial reflexes were also exaggerated.

About one month after admission the patient began to have, on an average, twenty or thirty attacks a day, which were generally convulsive, both sides being involved simultaneously in the onset, but the right side remained in

the tonic stage throughout. After the attacks had been as frequent as above stated for two days, the patient began to lose muscular power in the right side and at the end of the fourth day complete paralysis of that side obtained, which existed for an entire day; but when the attacks had diminished in frequency to fourteen in twenty-four hours, the patient recovered to a slight extent the lost power of the affected side.

Two days after this his attacks began to resume their former frequency and severity and the paralysis once more returned as severe and as complete as at first.

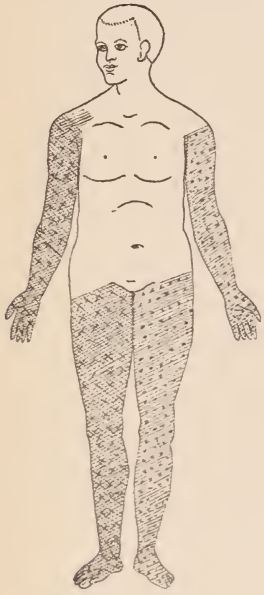


FIG. 35.

//=slight convulsion.
::=slight paralysis.

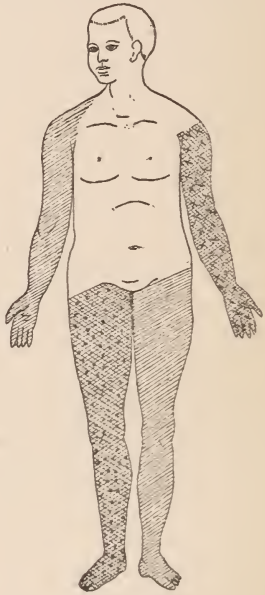


FIG. 36.

#=severe convulsion.
×=severe paralysis.

The left side also showed a diminished power of motion and co-ordination and was also probably somewhat affected by the general convulsions, but in a much less degree than the right side. (See Text-Figure 35).

None of the attacks were of the petit mal type; they were all severe and generally convulsive. In reply to a

letter of inquiry sent to patient's mother, she states that his previous functional paralysis, following epileptic attacks, existed in both legs and nowhere else in the body. He had suffered from but one attack of this kind of paralysis; it lasted two weeks and followed twenty-five severe seizures occurring in rapid succession in a single day. This was three years previous to his admission to Craig Colony. The paralysis was sudden in its onset and the recovery from it was steady and complete.

At present, January, 1897, considerable pulse elevation takes place after his series of grand mal attacks, ranging from 110 to 140. Temperature has reached as high as 102.5° F. To all appearances the patient suffers from true idiopathic epileptic seizures of grand mal type which are followed by a temporary localized paresis or paralysis of left side. The attacks begin as shown in Text-Figure 38, which is a posed figure of the patient presenting characteristic initial movement in convulsive period, followed by temporary complete left side paralysis.

All patient's attacks are sudden and violent; successive physiologic groups of muscles are involved in the tonic stage giving the patient the appearance of performing semi-purposeful movements as in hysteria. Consciousness, which is always lost as soon as the first muscular spasm ensues, returnes at once on cessation of spasm, if no more than eight or ten attacks occur daily. In the spasms the tonic states of muscular rigidity predominate. A few times he has had an aura which has invariably been a sharp pain in left wrist.

During the past year the character of attacks has changed and has remained constant without variation, to this new order. (He has had 2,500 during the year). Tonic spasm begins at once in all the muscles of the left arm, then crosses and passes to right leg, finally begins simultaneously in left leg and right arm. Spasms last for but a few seconds and are never clonic, even in series. Text-Figure 37 is drawn from photograph of an actual convulsion after it had become general, giving appearance of equal bilateral involvement. The muscles of left arm

and right leg were much more rigid than those of the right arm and left leg; the face was not involved and the patient retained consciousness. The original photograph is reproduced on Plate V, Figure 3.

Right knee-jerk and left wrist-jerk diminished immediately after seizure; in ten minutes after they become normal and are exaggerated in thirty seconds.

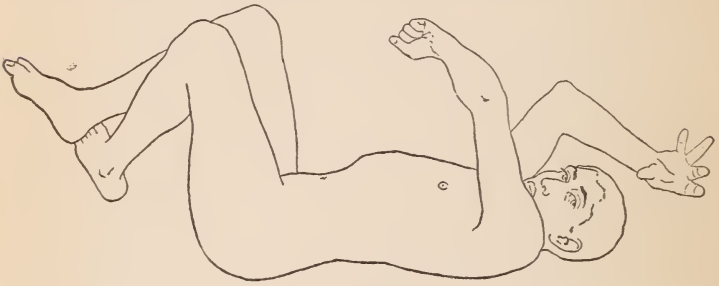


FIG. 37.

FIG. 38.

Slight paralysis is found after all these attacks singly; whenever a series of eight or ten occur in twenty-four hours, marked palsy is noticed; it is always in the left arm and right leg, most marked in left arm. Two attacks of status epilepticus during the year have rendered left arm and right leg hemiplegic for several days. Text-Figure 36 illustrates the crossed convulsive phenomenon and the sequent paralysis.

Right knee and left wrist-jerks have now become permanently exaggerated.

Patient died in status epilepticus August 27, 1898, from exhaustion after seventy-four seizures the same as above described, occurring in four hours.

Autopsy showed nothing unusual in brain or cord to explain local exhaustive phenomena.

The following case which I have given personal study for months is unusual in many particulars. While presenting many interesting phenomena of exhaustion-paralysis, it will be interesting to dilate at some length upon other symptoms of the case.

CASE 18.—L. B.; aged 15. Maternal great grandmother had epilepsy. The mother is neurotic, had periodic attacks of headache, and fainting spells (for one year) at the age of thirteen; maternal grandfather an inebriate. The father is of a very nervous and morbidly sensitive disposition.

Patient is the first child in family of two children. The younger, at present eight years of age, is very neurotic and has shown some hysterical symptoms. Patient was born at full term and delivery was normal. Dentition was easy and normal and nothing very unusual was noticed in patient's life until the thirteenth year. Her sleep in infancy (although there was a good deal of bodily restlessness at times) was always perfect, and she was never subject to wakeful periods. In her eighth year while at school, she had occasional periods of "tired, sleepy feelings" in the afternoon. At oral examinations she frequently failed because of physical fright. A thorough and most painstaking mental and physical examination proved for the most part, strictly negative and is omitted here.

At thirteen years of age patient began having daily attacks of complete flexion of right arm, forearm and hand, not painful, and she was unable to control these contractures. Before all of these attacks she has a well defined but indescribable aura, a peculiar sensation of weight or tension in the biceps of the right arm, traveling in a wavelike manner from head to foot. The aura is never felt in the trunk in its supposed progress from right

shoulder to right thigh. This aura disappears and reappears again and again, sometimes for a whole day about one or three minutes apart, ultimately ending in an attack. Face is hot and flushed whenever the aura appears. The patient can prevent the aura and postpone the attack for hours, but ultimately the convulsion appears. The method of inhibiting the aura most commonly used is to keep the hand and arm occupied in some manner when the peculiar sensation which she is not able to describe, appears. (Epileptics are frequently unable to describe the aura because it is not analogous to any known sensation; probably in this instance the prolonged aura is the beginning patho-physiological excess of cerebral impulses passing to muscles which are to be convulsed later). Frequently, after postponing the seizures for hours, the patient will declare she "can't be bothered any longer," and on lying down a series of convulsions will occur and she then gets up feeling assured that she will be free for some time from the troublesome symptoms. Whenever these warning sensations are felt she is certain to be attacked at the next period of rest in sleep, or when much fatigued. Severe attacks, which always occur at night, are frequently postponed for some time by the patient staying awake beyond the usual hour for their appearance (8.30 to 10.30 P. M.)

Attacks became more frequent in a few months from the beginning until at the end of six months they were occurring as many as twenty each night, followed by temporary paralysis of right arm for a few moments. The arm was more frequently "numb" than powerless. Patient has never shown any of the mental stigmata of epilepsy and is unusually bright mentally and very capable. The attacks have never materially interfered with her work by day.

In July, 1898, patient was sent to Craig Colony and has since been directly under my observation, and I have witnessed many hundreds of her attacks. Several specific descriptions of seizures with detailed attendant phenomena

will be given after general statement of the convulsions is set forth.

Hysteria, simulation of mild attacks, paramyoclonic phenomena rarely seen in epilepsy, have all been held in mind in the case study.

Usually patient is awakened out of sound sleep and while the convulsion is beginning calls for the nurse and directs her what to do. The convulsive movements generally begin in biceps, spreading up and down the arm and in a few seconds pass into the thigh muscles and then become general. The muscular spasm frequently produces slow gyratory movements of the extremities without pain or loss of power for voluntary acts. The spasm is most marked in proximal muscles of extremities and in the lighter attacks (about two-thirds of entire number) no clonic spasm occurs. Patient is often contorted in many peculiar ways and frequently laughs at her helplessness.

Frequently a breath of cold air from a window, or blowing upon right biceps, laid bare, will produce slight attacks quite analogous to the Case 17, W. S., in which the tapping for knee-jerks excites attacks. In the morning tests right hand pressure on dynamometer at 35 will just start the aura and a seizure follows if the effort is increased two points. At night, 25 or 30 will produce attacks.

All approved medication and methods which have been tried have no influence on attacks. Knee-jerks and wrist-jerks are lost on right side just after attacks, to become much exaggerated after a few minutes. They remain permanently exaggerated at present. A series of temperature and dynamometer tests were made in slight attacks, proving negative.

Probably the slight forms of spasm are in all respects akin to the severe variety, lacking only in intensity and completeness of nerve discharge.

The author cannot present the case more graphically than by presenting clinical notes covering observations of a series of attacks.

Patient sleeps by day to try breaking the night periods.

December 4, 1898.—Cold spinal douche 8.38 A. M.; slight attack 8.40, immediately after douche. Right forearm and lower limb above knees massaged for three minutes each—8.45, severe attack; 10.15, very severe; 11.20, slight; 12.05, 12.21, 1.14, very severe; 2.25, 3.30, slight. Bromide mixture (50 per cent) one teaspoonful at 2.30.

Complains of cold chills in the back, pain in head and lower limbs; general exhaustion phenomena. Temperature, 100; pulse, 80; respiration, 20. Just previous to attack at 8.40 patient saw bright flashes of light in the form of round balls, beginning with blue and running all through the colors; more of the blue and yellow than others; all passing from right to left.

Slight attacks, 9.16 P. M., 9.50, 10.25, 10.48, 11.50, 12.35, 1.25, 2.20, 2.58, 3.35.

Severe attacks, 12.16 A. M., 1.57, 5.20, 6.00.

Temperature.	Pulse.	Respiration.
101° F.....	108	30 at 7.55 P. M.
100° F.....	96	22 at 10.00 P. M.
99° F.....	88	20 at 2.00 A. M.

Severe attack 10.15 A. M.; patient awoke uttering a peculiar muttering sound. Shoulders elevated and head drawn down on chest. Shoulders were lowered and arms abducted from body. Right wrist was flexed toward body, fingers flexed at all joints in convulsive position. This position was maintained throughout tonic stage. The right hand was clenched with thumb flexed on the palm and fingers flexed over thumb; this position was maintained for a few seconds. Then as the spasm became more severe the left arm was moved about violently and patient grasped frantically with hand in an effort to hold something to stop the gyration of the arm. The lower limbs were held in rigid position, ankles inclined inward

and toes in hyperextension. Feet crossed at ankles. Face cyanosed, eyes turned upward, pupils dilated. Patient passed into a clonic stage; froth from mouth. Urine was voided after clonic stage ended. Consciousness lost at end of clonic stage. This clinical description of a fit is quite typical of a grand mal fit; it was not followed by any local exhaustion phenomenon.

Examination for indican showed its marked presence in five tests December 2d, 3d and 4th, being most marked in morning tests. The condition was not noticed after the last date given above and it probably played little or no part in the causal element.

Temperature.	Pulse.	Respiration.
98½° F.....	112	24, 10 min. after attack.
68° F.....	104	22, 30 min. after attack.

December 5th.—Patient had a similar attack as that described December 4 and it was followed by fibrillary tremor in right fingers. There was a tired feeling in right arm with dull ache in biceps. See Text-Figure 39.

December 17th, severe attack at 5.40 A. M. Began in the usual manner described, followed by tonic and clonic spasms. Congestion of face, froth from mouth, and stertor. Consciousness lost at end of clonic stage. Patient lay in a comatose condition for a short time and gradually relapsed into a sound natural sleep. Numbness of right arm (sensory type of paralysis); fibrillary tremor in thumb and first finger (mild motor paralysis). See Text-Figure 40.

December 17th.—Warm bath followed by massage of arm at 8.30 P. M. Severe attack 9.30, 10.30, 11.15, 11.45, 12.05, 2.00, 2.45, 3.30, 4.40, 5.40.

The first eight attacks were of usual conscious character. That at 4.40 began in the usual manner, followed by relaxation of the muscles, then followed intervals of spasmodic, jerky respiratory acts (clonic spasm of diaphragm). Muscles relaxed for a few seconds and then gradually contracted on right side, while apparently the left limbs were motionless and not convulsed. Right arm

flexed with elbow out, palm of hand turned inward, while the fingers showed marked extension. The same position was noticed in the right foot and leg. Sole of foot turned inward, with toes in hyperextension. Head drawn to the right also. Frothed from mouth in stertor. Seizure lasted 2½ minutes. Slight paresis in arm with tremor for twenty minutes; tired feeling in right leg for three minutes. Here we see that second seizures predominated in

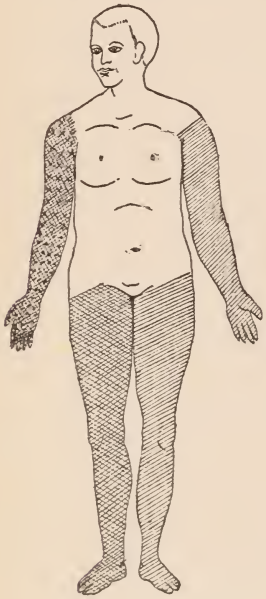


FIG. 39.

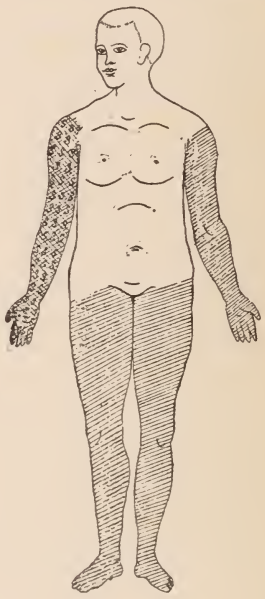


FIG. 40.

//=slight convulsion. #=severe convulsion.
::=slight paralysis. ×=severe paralysis.
S=loss of cutaneous sensation.

the right side and occurred so soon that the previous general exhaustion from a fit could not be recovered from and as a consequence a local paralytic state followed.

December 18th.—Usual bath and massage at 8.30 P. M. Slight attacks at 7.40 and 8.15. Severe at 9.30, 10.25, 10.40, 12.15, 12.25, 4.50, 5.40.

Attack at 4.50 began in the usual manner, followed

after one minute by a general tonic spasm for one-half minute; feet inclined inward with toes in hyperextension; arms straight by the sides in complete pronation with fingers flexed. At the expiration of half a minute the muscles of the right side relaxed, head drawn to left side with chin resting on shoulder, mouth open and drawn to left. Eyes turned to the left in a fixed stare; pupils much dilated. Patient remained in this condition 1½ minutes, the face apparently retaining its normal color. Then the muscles gradually relaxed (without clonic spasm) and patient lay in a comatose condition for a short time, gradually falling into a sound natural sleep. Paresis of right arm; tremor in left hand for two minutes.

December 21st.—Warm bath and massage at 8.15 P. M. Slight attack at 8.30; severe 9.00, 10.15, 10.45, 12.30, 4.15, 5.30, 6.50.

Attack at 4.15 began in the usual manner and continued so for one-half minute, when the head drew downward and to the left side. The eyes and the mouth were also drawn to the left. Eyes kept up a continuous winking motion. Nostrils widely dilated; patient making a snorting sound. Tonic stage lasted only a few seconds and was succeeded by a mild clonic spasm, lasting about twenty seconds, leaving the patient in an exhausted condition, but consciousness retained. Sound natural sleep soon followed. One hour and fifteen minutes afterward patient was awakened by a general typical epileptic convulsion of tonic and clonic stages; consciousness lost from the start. Patient was much exhausted during forenoon but no local paralysis resulted.

December 24th.—Severe attack at 4.50 A. M. Began in the usual manner, lasting one-half minute. Then general clonic spasm followed. The head was drawn downward and to the left. Mouth and eyes also drawn to left side. Face congested; froth from mouth. Clonic stage lasted one-half minute; stertor five minutes, patient gradually falling into natural sleep. This seemed as distinctly epileptic as the one on the 21st; consciousness lost from the start. No paralysis was found.

January 2, 1899.—Slight attacks 7.30 P. M., 11.40, 12.15. Severe attacks 8.40 P. M., 10.05, 11.15, 1.25, 2.07, 5.30.

Attack at 2.07 began in the usual manner, continuing for one-half minute. Then the head was drawn to the right and downward until the chin touched the shoulder. The mouth and eyes were drawn to the right side also. Face much congested. The lower limbs were in anatomical position with the ankles slightly curved, the feet inclined inward; toes extended. In the effort to prevent convulsion the left arm was voluntarily straightened out by the side and the hand clenched. Right arm was flexed at the elbow and wrist and the hand clenched tightly in convulsion. Tonic spasm was succeeded by a slight clonic spasm lasting about half a minute. Consciousness was retained during attack and was perfect. Patient was much exhausted and gradually fell into natural sleep.

January 3d.—During the night patient had slight attacks at 9.45, 10.45 and 11.05 and severe attacks at 9.20, 12.40, 4.20 and 5.50.

Attack at 4.20 began in the usual manner, only in a lighter form, and lasted only a few seconds. The muscles quickly relaxed and the whole body became limp, accompanied by violent trembling in the whole right side lasting 1½ minutes. The eyes were closed and the face very pale. Respiration difficult. Consciousness was retained throughout. Right arm was paralyzed for three minutes after attack and was then raised with great difficulty. Tactile and pain sense diminished. See Text-Figure 41.

January 4th.—Severe attacks at 9.45 P. M., 5.20 and 6.15 A. M. Slight attacks at 10.40, 12.00, 1.10 and 2.30.

In the beginning of all the slight attacks and continuing throughout some of them, physiological groups of muscles were successively involved but in no definite order, producing semi-purposeful movements. These movements are simply gyrations of the extremities in an arythmic manner, what might be defined as a general mobile spasm, presenting, in some ways, remarkable resemblance to true chorea.

The severe attacks began in the usual manner with eyes

open and turned to the right. Patient remaining in this condition for thirty seconds. Then the head was drawn downward until the chin rested on the right shoulder, the mouth partly open and drawn to the right also. Fingers flexed on palm with thumbs protruding through first and second fingers. Arms straight by sides. Feet inclined inward from ankles, with toes of right foot overlapping those of the left. Limbs straight and close together. Face deeply congested; lips pale. Tonic stage lasted two

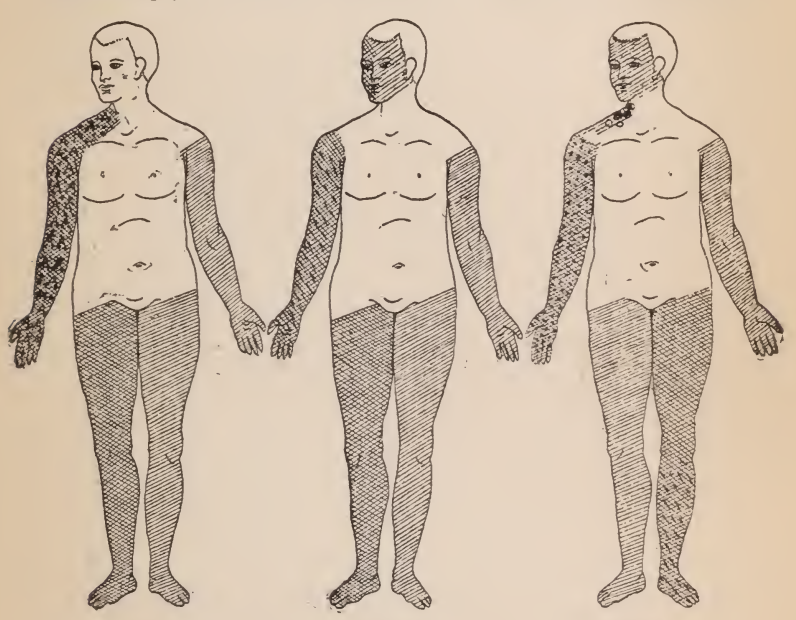


FIG. 41. FIG. 42. FIG. 43.

//=slight convulsion. #=severe convulsion.
O=contraction. ,',',' =tremor.
A=paræsthesia and anæsthesia.

minutes, during the latter part of which the eye-balls closed and twitched rapidly. Muscles relaxed and a general clonic spasm followed, lasting for thirty seconds. Stertor lasted six minutes, during which time the face resumed its natural color. No paralysis but marked tremor

for four hours in right arm. Patient could not speak during the seizure because of right hemi-lingual spasm. Three minutes after the attack she related, with minutest detail, the things which occurred during the seizure. See Text-Figure 42.

January 6th.—Patient had a severe seizure at 4.30 A. M., which began in the usual manner. After thirty seconds the eyes and mouth turned to the right; a few seconds later the head also turned to the right. Right arm was straight by the side with the wrist in normal position and fingers flexed. Right leg was straight, with foot inclined inward from ankle, toes extended. The left leg was relaxed. After one minute the muscles of the right side relaxed and a slight clonic spasm followed, confined to right side.

Patient was conscious during seizure and did not relapse into a stupor afterward. She was unable to raise right arm for about one minute after seizure, although sensation was retained.

January 11th.—During the night patient had slight attacks at 10.15, 12.20, 1.10 and 2.15. Severe attacks at 2.00, 4.10, 5.15, 6.00 and 6.58. The attack at 4.10 began in the usual manner, but at the clonic stage patient uttered loud screams. After a few seconds the eyes rolled to the left. The head and body turned slightly to the left side. Both sides were equally involved in all respects. Limbs straight, wrists flexed, hands clenched, feet inclined inward from ankles, toes extended, face congested, pupils and nostrils dilated, mouth open, patient making a snorting sound, respiration very difficult. Gradually muscles relaxed, eyes and mouth closed and a general clonic spasm followed. The muscles of the eyes and mouth were apparently not convulsed during clonic stage. Duration of the tonic stage, one minute; of the clonic stage, two minutes, and of stertor, two minutes. The right arm remained paralyzed for 2½ minutes after stertor. Sensation was not lost nor apparently dulled. Patient remained conscious throughout attack but was unable to speak for two minutes afterward, during which time she looked about attempting

to talk (temporary aphasia). Speech returned slowly and was thick for three hours. Patient remembered having had her arm rubbed by the nurse during the seizure and repeated conversation and questions.

January 12th.—Patient had slight convulsions at 7.52 P. M., 8.55, 10.30 and 12.05. Severe seizures at 9.25, 1.35 and 5.20. The onset of attack at 5.20 was different in character from the usual onset in severe convulsions. The head was drawn downward with chin resting on right shoulder. Hands clenched with thumb protruding between first and second fingers, wrist and elbow flexed, arm thrown upward over the head, lower limbs abducted, knees flexed, foot inclined inward from ankle, toes flexed. The feet nearly touched the head; the body was in such a position that the upper and lower extremities were in close proximity. The patient then uttered loud screams. At the end of thirty seconds the limbs and body were straightened. The hands and feet maintained their attitude except that the toes were changed from flexion to extension. Invasion on left side began in the foot. Tonic stage, one minute; clonic stage, two minutes; stertor, about 2½ minutes. Consciousness was retained throughout convulsion. Patient lost control of voice but made partially successful efforts to control scream; she did not get control, however, until four had been given. There was marked tremor and tired feeling in right arm.

January 22d.—Severe convulsion at 3.50 A. M. began in the usual manner and varied from other severe convulsions only in the order of invasion. The right arm and both lower limbs assumed tonic stage one-half minute before the left arm. During the first few seconds of the seizure the left arm was waved about in the air and was gradually drawn down by patient's side and assumed tonic stage at the expiration of thirty seconds, passing through convulsion when the rest of body had ceased. Paralysis for thirty seconds in right arm; tremor in left foot. During the attack the head was drawn to the left side with face looking to the right. The body from the waist upward was twisted to the left. The face and neck were con-

gested and the neck muscles remained rigid on right side for three minutes after the attack. Tonic stage, one minute; clonic stage, two minutes. Patient spoke with great effort in three minutes after convulsion; repeated all that had occurred during seizure. Tongue was protruded with difficulty, showing hemi-lingual spasm of right side persisting for a few seconds. See Text-Figure 43.

February 6th, patient had severe attack at 3.30 A. M. The right arm and both lower extremities were involved in tonic spasm one-half minute before the left arm began to be convulsed. The left arm continued in spasm for one-half minute after the convulsions in right arm and lower limbs had ceased. Tonic stage lasted 1¼ minutes. Only three or four clonic movements; no stertor or coma followed clonic stage, but exhaustion was quite complete. The patient was aroused by shaking. Pain and tactile sense considerably diminished for thirty seconds. Postural sense was retained. Right hand and arm remained in tremor for two minutes. A "tired dull ache" continued half an hour.

Patient has premonitory signs for several hours before seizure. At such times irritation of the area on the arm shown in photograph Plate V, Fig. 1, will bring on an attack at once.

The act of tickling this area with the fingers produces an attack if the sensation of a threatened seizure has been present during the day; it is quite as powerful a causative agent in the production of seizures as extreme pressure or more irritating stimuli. Pin pricking is slower in its action of producing the attacks. Pressure on the arm by the hand grasp does not cause an attack as soon as a rapid, light, intermittent pressure; this last form of stimulation or irritation typifies the best manner of producing attacks. Rapid intermittent stimulation appears to send an excessive number of distinct shocks to the right arm centre and immediately a seizure appears. Many attacks have been produced or hastened by having the sleeve of the shirt waist strike against the arm from the pressure of a slight breeze. Passive movements of flexion, extension, abduction or

adduction are all about equally apt in causing a seizure. On the contrary, active movements of a slow, rhythmic character discourage the presence of seizures. Warm air blown upon the arm will cause attacks to appear in about one or two minutes. Cold air from ether spray, or applied ice, brings an attack immediately, especially if applied intermittently. The production of attacks by dynamometric test has already been mentioned. The attacks produced are not different in character or degree from those heretofore described when special stimuli have not been applied. They are frequently grand mal in character but never attended by loss of consciousness, although deep respiration attended by stertor is present for a few seconds after.

This patient (L. B.) states that at all times when the muscular movements are general and apparently equal over the entire body, the convulsion is really most intense or "harder" in the right arm, right leg next, and least in the left leg and left arm.

It would seem that the somewhat circumscribed area first involved compelled or induced different and fairly stable centres to co-operate with it in the disease phenomena. A speculative analysis of the cause and lesion in the left biceps centre giving rise to the epileptic condition might prove interesting. An equally important study in this case might be made upon the relationship of consciousness and epileptic convulsions in their various alterations. A highly suggestive fact lies in the observation that in almost all cases showing paralytic symptoms of an exhaustive nature the retention or loss of consciousness is not apparently constant even when partial seizures occur. When attacks are general in such cases, still disorders of consciousness hold to many distinctive peculiarities. Disorders of consciousness are not entirely parallel with areas of diffusion or length of muscular spasm. It would seem to rest largely in the kind of muscular movements. Neither is location of the

initial muscular spasm to be ignored in such study; proximal muscles of the extremities, involved first and most, favor the retention of consciousness. Such facts of observation are not altogether without foundation in recognized physiological laws of cerebral dynamics.

It is very interesting to observe the mild form of paralysis seen in the "tremors" and "tired feeling;" probably the disappearance of the latter marks a nearly complete inter-paroxysmal restoration of power. These conditions of tremor and "tired sensations" accord well with physiological and pathological facts in exhaustion theory.

We would call especial attention to the fact that the complete restoration of motor power is successively less completely recovered from as the disease persists; the nervous elasticity of the centre is less perfect. This phenomenon is quite similar to other cases recorded by Gowers, Féré, and Dutil, who showed that repeated exhaustion finally ended in anomalous hemiplegia.

There seems to be no reason why we should not infer that these lighter grades of exhaustion sequelæ, tremor and tired muscular feeling, (the former seen in 75 per cent of all epileptics, Raymond; the latter covered by coma Jackson), are closely allied to general exhaustion after general seizures.

We consider this case * a fair illustration of that rare type of epilepsy associated with the so-called epileptogenic zone; the area is distinctly circumscribed, sensory and motor irritation produce attacks, but in all instances (in

* November 10, 1899, L. B. has had no convulsions for seven months. She takes 480 gr. of potass. brom. daily, and has shown remarkable physical and mental improvement. Epileptogenic zone is suppressed and right hand has normal power.

December 15, 1899, epileptic attacks are reappearing again, presenting the usual symptoms.

this case as well as the other two cases I have seen) there was a sort of preparedness in the part of the cortical centre producing epileptic phenomena. Many and varied means have been used to cut short an attack when once begun; they have all been futile. On rare occasions the patient has been able by strong volitional effort to control the left arm and still more rarely, the left leg. She stops the arm in tonic or clonic stage by directing the extremities to a definite purposeful movement. Paralytic phenomena of right arm is unchanged in produced attacks.

It has been proven to my satisfaction and to that of my colleagues, that, although representative of epileptogenic zone cases, the real cause in this case lies in the left motor area and especially in the centre for the arm.

It is not necessarily the most movement produced, it may be the most energy liberated from unequally charged centres that cause paralysis; thus some particular centre fails in discharging even its lessened function without suffering in manifest manner in the after exhaustion-paralysis.

In proof of the fact that the exhaustion of central nervous elements may take place with but little muscular convulsion, we would quote the well known physiological experiments that demonstrate that a nerve tires long before the muscles. The nerve requires a correspondingly longer time for recovery. Bernstein's experiments (Landois and Stirling, page 587, ¶ 325) seem to prove these statements. He also found that the same principles were applicable to the sensory nerves. Both clinically and by experiments it has been found that a nerve recovers from fatigue first slowly, then rapidly, and finally slowly. Degenerative changes take place more rapidly

in a nerve in which recuperation is delayed for any length of time.

Two cases of temporary exhaustion in which many successive exhaustions have occurred have recently come to autopsy and I expect that careful pathological study of the entire motor tract will prove exceedingly interesting.

Case 19.—F. D., female, age 13; epilepsy began at 2½ years of age. Father intemperate and epileptic. Patient's attacks have always been of grand mal type of classic epilepsy.

Admitted to Craig Colony, October 27, 1898. During the month of November patient had twelve attacks; in December six; in January, 1899, twelve. All of these attacks were severe.

On February 13 she had from 6 A. M. to 2.40 P. M. sixty-two convulsions, the last followed by status. These convulsions were followed by left hemiplegia of an exhaustive character.

The convulsions began on the left side of the head, then the eyes and head were drawn to the left; a few seconds after the left arm and hand were involved, then the left leg. The fingers of the left hand took up the common convulsive position of epileptic paroxysms. The arm and leg remained extended, in a straight line with the body through the tonic (five seconds) and clonic (fifteen seconds) periods of the seizures. There was no frothing, nor stertor, and the tongue was not bitten. The right arm and right leg, which remained unconvulsed during the first sixty-two attacks of this status period, were frequently thrown about the bed after attacks, but the left side remained without motion.

Sensation seemed to be present throughout, as the hypodermic injection on the left side was received with moaning and much movement of resistance on the non-paralyzed side. No unusual appearance was presented by the skin of the paralyzed side.

The coma during the first sixty-two attacks was never

so severe or prolonged but that it was possible to arouse the patient, consequently the general exhaustion could not be counted severe. Temperature quickly ran to 103.4° F., pulse 140, and respiration 32.

During the few minutes' respite, after this series of convulsions, two ascaridæ lumbricoides were expelled from the mouth, and a few seconds later a general convulsion ensued. Partial motor power returned to the left side immediately after this general fit. All deep reflexes were abolished and remained so for several days.

From 4.40 P. M. until 7.10 the next morning, 142 severe general fits occurred, but the degree of paralysis on the left side became less marked all the time. The condition was now typical of status in every respect. At one time the temperature was 107.4° F. Patient made a slow recovery from status in two weeks. Many evidences of left hemiplegia were still present in this case for the three months of convalescence following this status period. Text-Figure 44 drawn from a photograph (Plate III, Fig. 1) of the patient taken two months after the condition appeared, shows the left hemiplegic state and rigidity of left hand still unrecovered from. Patient was recovering rapidly from this exhaustive state when the next series began in which patient died. The persistence of this state caused much doubt as to the original condition being purely exhaustion, but the autopsy findings cleared the ground for exhaustion. The persistence of the left hemiplegia from the exhaustive condition, can be well observed from the following hand grasps shown in the dynamometric test.

DYNAMOMETRIC TEST.

April 6—Right	38	Left	30
7—Right	34	Left	32
8—Right	38	Left	28
9—Right	32	Left	30
10—Right	45	Left	38
11—Right	38	Left	20
12—Right	44	Left	38
13—Right	38	Left	30
14—Right	40	Left	36
15—Right	36	Left	28
16—Right	42	Left	34

May 23, patient began having many serial attacks of grand mal type, and soon passed into status again, and died from exhaustion and pneumonia, five days after. She had 403 severe convulsions in this series.

Although the attacks were all general (not partial at any time) the left side suffered noticeably in paralysis.

Her attacks were always about as follows, noted on May 25, 1899: Attack began with muscular convulsion about the left eye and left corner of mouth, passing to left arm, left leg, foot in position of Pes Calvus. Convulsion was tonic for a period of a few seconds only. The remainder of movements on the left side were clonic. After about five seconds the right leg became involved distally, the left arm distally, and finally, the order of invasion was completed by the right side of face becoming involved, the same as left, at the beginning. The convulsion was clonic throughout on right side, and generally persisted for three or four seconds longer on left side, and the convulsion was by far the severest on the left side.

Dermographism gradually became more marked as attacks became more frequent, thus proving the vasomotor exhaustion, the most trustworthy evidence of critical general exhaustion in status epilepticus.

After second attack, post-epileptic contracture, so-called paralytic equivalent was found in right arm. Temporary paralysis of entire left side was present for a few seconds after each attack. See Text-Figure 45. Diffuse papillary erythema was present on the face after convulsion. The left hand was cyanosed.

May 26, convulsions began the same as yesterday in left side of face, left arm, etc. Internal strasbismus of left eye. Order of invasion alternates with left eye and left corner of mouth, neither one constant. When the left arm became involved it underwent external rotation, flexion, abduction, extention, internal rotation, then tonic rigidity. Reflexes were not present in interparoxysmal state, but during the tonic convulsion they were present in both extremities. The exaggeration

of the knee-jerk could be obtained and was proportionate to the severity of the muscular convulsion.

In order to determine whether or not the paralysis in this case, present in the left side during status, was really on the side most convulsed, observations were carefully made of the rate of muscular invasion in the different parts of the body.

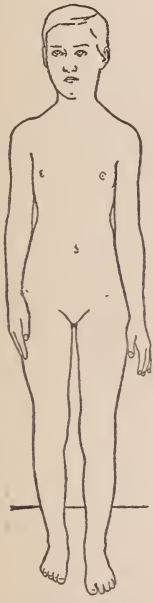


FIG. 44.

//=slight convulsion.
:: =slight paralysis.

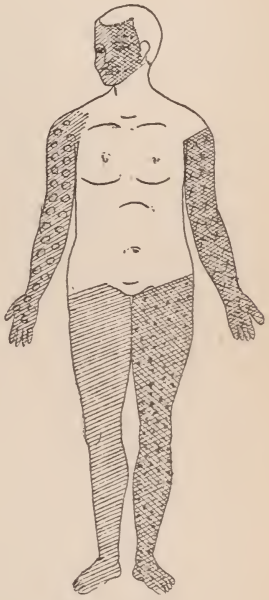


FIG. 45.

#=severe convulsion.
×=severe paralysis.

O=contraction.

Length of time elapsing between the invasion of different parts of the body during a severe convulsion: 20 seconds after left side of face became involved, left arm became involved; 5 seconds later, left leg; 25 seconds later, right leg; 5 seconds later, right arm; 25 seconds later, right side of face.

Duration of convulsion in different parts of the body,

second involvement of left face is to be noted. Left side of face first, 55 seconds; second, 1 minute 15 seconds; left arm, 35 seconds; left leg, 32 seconds; right leg, 35 seconds; right arm, 44 seconds; right side of face, 44 seconds. Although right face and arm were involved for a long time, they were for the most part in clonic spasm and did not proximate the left side in real severity of convulsion.

The photographs of this case are of unusual interest, and add additional proof to the fact that the paralysis is most prominent in parts most convulsed. Text-Figures 46 and 47, drawn from photographs (reproduced on Plate

FIG. 46.

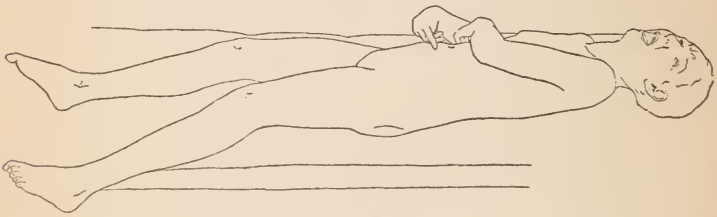


FIG. 47.

III, Figs. 2 and 3) of fits, present in a graphic manner the description of the fits just mentioned in the preceding paragraph. Fig. 46 shows patient after the initial spasm, which was not very severe, had passed over to the right side; Fig. 47 shows the *second* involvement of the *left side* where the convulsion began and ended.

The second muscular involvement of left face in the same convulsion is very unusual, and affords a very tempting field of speculation in regard to the areas of cortical discharge.

An autopsy was held two hours after death: Brain

and membranes were to all macroscopic appearances normal, except the brain tissue was of ivory whiteness; no hemorrhages, clots or local areas of softening were found. The brain in all respects was well developed. A broncho-pneumonia of right lung was found. Spleen was large, soft and friable, otherwise normal. Ten ascaridæ lumbricoides were found in small intestine. Microscopic examination has not been completed and cannot be presented at this time.

This case of F. D. is only one of many militating against the Jackson theory, that in status, the exhaustion runs into or is covered by coma.

We have seen that almost all hemiplegic epileptics in serial attacks or status, present local exhaustion phenomena in paralyzed parts, independent of the true coma and general exhaustion, which latter condition all status cases invariably show.

Chapter XI.

GENERAL CONCLUSIONS.

Although having expressed my views freely all through the monograph, I feel warranted in drawing the following deductions from this study in epilepsy:

First.—The theory of exhaustion-paralysis is conclusively proven by physiological experimentation and especially by pathological data derived from the observation of phenomena in epilepsy.

Second.—Exhaustion-paralysis is localized to parts participating in the local spasms, or confined to those parts most convulsed in general seizures. Although there may appear to be exceptions to this general rule such cases will always be found to follow the rule if carefully observed.

Third.—The temporary paralysis may become perma-

nent and exist as a true hemiplegia with organic changes of a varied nature.

Fourth.—It is not necessary to invoke any other state than exhaustion to explain the temporary paralysis in epilepsy.

Fifth.—Temporary exhaustion-paralysis is essentially an exhaustion of cerebral centres and the apparent severity of muscular convulsions is not a fair index to the amount of paralysis that may follow.

Sixth.—True exhaustion cases independent of infantile palsy affections are not common. The differential diagnosis of the two lesions or states is extremely difficult but most important, because the exhaustion cases independent of organic lesions help bridge over the wide breach existing between so-called Jacksonian epilepsy and idiopathic epilepsy.

Seventh.—Researches upon the disorders of motility which partake of the nature of exhaustive phenomena following convulsions, as seen in epileptics, seems to suggest some noteworthy points. First, that the degenerative process of exhaustion-paralysis, as manifested in epilepsy, is closely allied to that of ordinary paralytic states. In epilepsy, when no real paralysis exists, many disorders of motion are seen, such as slowness in movements, tremors, irregular and inco-ordinate bodily movements in purposeful acts. Due allowance must be made for the fact that the epileptic brain, in the great majority of instances, is much incapacitated, and the dynamic flow from its motor centres is never perfect, thus paving the way for its further motor degeneracy seen in various paralytic states.

With this fact in mind, we should not charge to the account of epilepsy as a cause more than that which may

be due to a previous disability of the motor centres. The great frequency of epilepsy as a symptom of gross cerebral lesions of a paralytic nature, and its close relationship to other spasmodic and periodic diseases of the nervous system which have paralytic phenomena lends material aid to our clinical hypothesis of a close association of epilepsy and paralysis.

To the speculative clinician many data are here presented for drawing conclusions of *epileptic foci of a paralytic nature* in the brain. The so-called mixed aura in which widely different portions of the brain are simultaneously involved, and only in the early stage of epileptic paroxysm, give further proof of the fact that different anatomical and physiological parts of the brain may be severally involved; finally, minute disseminated patches of softening going on to sclerosis in different areas of the epileptic brain, so repeatedly demonstrated, help on our hypothesis of epileptic foci in a striking manner.

Eighth.—We desire to call attention to the ineffectual methods of passive movements, massage and ordinary rubbing, to cut short or prevent epileptic muscular spasm. Indeed, in not a few instances, by passive exercise, we have been able to produce seizures. Undoubtedly the idea received much encouragement from the earlier notion that seizures began peripherally, and the muscular apparatus was primarily responsible for the convulsion, as seen in some of the other functional neuroses.

REFERENCES.

1. Strümpell: Text-Book of Medicine, page 732.
2. Dercum: American Text-Book of Nervous Diseases, pp. 296–315.
3. Osler: Practice of Medicine, p. 1006.
4. Ormerod: Diseases of the Nervous System.
5. Hirt: Diseases of the Nervous System, pp. 532-560.
6. H. Higier (Warsaw): Neurol. Centralblatt, 1897, p. 152.
7. H. Higier: Deutsche Zeitschr. f. Nervenheilkunde, 1899, (XIV) p. 325.
8. J. W. McConnell: Journal of Nervous and Mental Diseases, June, 1899.
9. T. M. Shaw: Ind. Med. Gaz., December, 1890.
10. R. T. Williamson: London Lancet, 1897, Vol. II, p. 1522.
11. Handfield Jones: Functional Nervous Disorders, London, 1880, p. 129.
12. Richer: L'hystéro-épilepsie, 1881, p. 552.
13. Hughes-Bennett: Brain, Vol. VII, p. 492.
14. Frank-Smith: Lancet, 1869, Vol. I, p. 427; British Medical Journal, 1876, Vol. II, 591, and 1874, Vol. II, 551.
15. Landois: Uræmie, 1891, page 153.
16. Rego: (Cited in Canstate Jahrb, 1842).
17. Dornbluth: (Ibid).
18. Lequinie: Belgium Acad. of Med., 1856.
19. Dunin: Berlin Klin. Woch., 1889.
20. Franck: Proceedings, Society of Biology; Gazhebdom, Nov. 8, 1878.
21. J. M. Bramwell: Brain, 1896, p. 91.
22. Braid: Original work upon hypnotism, published about 1845.
23. Briquet: Traité de l'Hystérie, 1859, p. 444.
24. Jaccoud: Les paraplégies, etc., 1864, p. 353.
25. Voisin: Épilepsie, 1897, p. 303.
26. Oppenheim: Nervenkrankheiten, 1898.
27. Féré: Twentieth Century Practice, 1897.
    Épilepsy et Épileptiques, 1890, p. 166.
28. Echeverria: Epilepsy, 1870.
29. Fournier: Syphilis du cerveau.
30. Ross: Treatise on Diseases of the Nervous System, Vol. II, p. 929.
31. Bravais: Thesis on Hemiplegic Epilepsy, Paris, 1827.
32. Todd: Clinical Lectures on Paralysis, etc. Lecture XIV, on Epileptic Hemiplegia, pp. 284-312.
33. Robertson: Edinburgh Medical Journal, Vol. XV, p. 513, 1867.
34. Gowers: Text-Book on Nervous Diseases, Vol. II, p. 743, 1893.
35. Gowers: Monograph on Epilepsy, 1881, p. 98.
36. Jackson: Brain, 1881.

37. Dutil: Soc. de Biol., Oct. 26, 1878.
38. Frank and Pitres: (Cited by Dutil in Soc. de Biol., Oct. 26, 1878.
39. Lowenfeld: Arch. f. Psychiatrie, XXI, 1890–1.
40. Bravais: Sur les symptomes et le traitment de l'épilepsie hémiplégique, Thèse de Paris, 1827.
41. Todd: Clinical Lectures on Paralysis, etc. Lecture XIV, on Epileptic Hemiplegia, pp. 284–312.
42. Robertson: Edin. Med. Jour., Vol. XV, 1867, p. 513.
43. Jackson: Brain, 1881.
44. Gowers: Epilepsy and Other Chronic Convulsive Diseases, etc. London, 1881, p. 98.
45. Charcot-Pitres: Case 3, published by Hitzig, Archiv für Psychiatrie und Nerven-Krankheiten, 1872 (Ueber einen interessanten abscess der Hernrinde).
46. Féré: Paralysis by Exhaustion, Brain, Vol. XI, pp. 208–215; Épilepsie et Épileptique, 1890, Obs. VI; Notes sur des attaques paralytiques chez un épileptique, Compt. rend. Soc. de Biol., 1896, p. 679.
47. Dutil: Des paralysies postépileptoides transitoires, Rev. de Med., 1883.
48. Russell: British Medical Journal, 1867, I, p. 732.
Medical Times and Gazette, Oct. 31, 1863.
Medical Times and Gazette, May 25, 1867.
49. Mercier: Brain, Vol. VI, p. 191.
50. Woodbury: Medical News, Philadelphia, 1895, LXVI, p. 462.
51. Blaud: Biblioth. med., t. LXII, 1818, p. 145. (Cited from Laudouzy, q. v).
52. Pereboom: Nova acta curios, nat. t. III, p. 20, (Féré, p. 19).
53. Charcot: Leçons sur les maladies du système nerveux, 1877, Vol. II, p. 343.
54. Grasset: Localisation dan les maladies cérébrales, 1880.
55. Pavy: Medical Times and Gazette, 1880, Vol. II, p. 536.
56. Sturges: (Date and reference unknown; cited by Ross, q. v).
57. Echeverria: Epilepsy, 1870.
58. Alder: Neurologische Centralblatt, 1898 (17), p. 691.
59. Comby: Arch. d. méd. d. enf., 1898, Vol. I, p. 227.
60. Fournier: Syphilis du cerveau, p. 240.
61. Hughes: St. Louis Courier of Medicine, 1882.
62. Bourneville: Icon. photo. de la Salpêtriére, 1878.
63. Prichard: Diseases of the Nervous System, Chap. on Partial Epilepsy, London, 1822.
64. Beevor: London Lancet, 1882, Vol. I, p. 307.
65. Russell: British Medical Journal, 1867, 1, p. 732.
Medical Times and Gazette, Oct. 31, 1863.
Medical Times and Gazette, May 25, 1867.

DESCRIPTION OF PLATES.

Plate I.

Figure 1.—Shows patient J. P. (Case 4) in interparoxysmal state. The exhaustion-paralysis is becoming more or less permanent, (patient has had 1500 attacks during the past year). Attention is called to the appearance of the left shoulder and hand, which are parts most persistently exhausted in seizures. They assume a position nearly similar to parts in old organic hemiplegia. No atrophy is manifested in left side and the muscles are still quite firm to the grasp. The muscles of left shoulder appear even hypertrophic; but this is not really so. The appearance is due to the slight hypertonicity which is now more or less persistent in all muscles of left side, due to persistent exhaustion.

Figure 2.—Photograph of J. P. shows patient in one of his usual grand mal seizures. Complete consciousness is retained, Convulsion is most severe on the left side, the left arm being more strongly involved than the left leg, as shown in the photograph by the prominence of the muscles in the arm, where the condition is tonic, while the convulsion in the leg is clonic, as shown by the blurred outline of left knee.

Figure 3.—A photograph from a hand posed, presents more clearly the position of left hand in Figure 2.

Figure 4.—(Posed) shows position of right hand of Figure 2. The marked contrast between pathological and physiological contracture is seen in Figure 2, as compared with posed figure in case F. R. given in text, (p. 414).

Plate II.

Figure 1.—C. P. (Case 5). An idiopathic epileptic of excellent physique. Photograph taken between the two periods of status mentioned in text. A previous exhaustion has been entirely recovered from.

Figure 2.—Shows the patient in general convulsion, in which the right arm and left leg are most convulsed. Muscles of left thigh (quadriceps femoris), left calf muscles and muscles of right arm and shoulder show this fact clearly. Left arm and hand are but little

involved. Head is rotated to the left and left side of neck is more involved than the right.

Figure 3.—Shows patient in stertor period immediately after the convulsion shown in Figure 2. The right arm and left leg show "contractures," the so-called post-paroxysmal paralytic equivalent. Attention is especially called to the muscles of right arm and left thigh, and to the complete inversion of left foot, which all show the muscular rigidity that was manifest in this case for a few seconds after the fit, illustrated in Figure 2.

Plate III.

Figure 1.—Case F. D., (Case 19), shows patient two months after first left side exhaustion-paralysis from status described in the text, and two weeks before the status which ended in death, one convulsion of which is well shown in Figures 3 and 4. The left side, and especially the left hand, still shows paralysis.

Figures 2 and 3, Case F. D. at two, of three periods of the same convulsion. (The photograph of the initial left side convulsion (the first period) which preceded the conditions shown in Figures 2 and 3 was too imperfect for reproduction).

Figure 2.—Was taken at the time, (second period) that the convulsion began to involve the right side of the body in tonic spasm. (Clonic element was never prominent in the case). Right side of face is in extreme spasm and the right arm and leg are very rigid. Attention is called to the position of right hand, a position as common in epileptic convulsion as the so-called "convulsive" or "clenched" hand. The muscles of entire left side are now flaccid, convulsions having ceased as soon as right side became involved.

Figure 3.—(Third Period). Shows the condition following on that shown in Figure 3 and illustrates the secondary and severe involvement of the left side (side first and most engaged in the fit). Convulsions about left face and neck are especially marked. Partial extension of left arm and flexion of right leg took place at the beginning of this secondary involvement of the left side. Attention is called to position of right hand in photograph, Figure 3; although flaccid now, it retains the mold of its former convulsed state, a condition common in cases showing exhaustion-paralysis.

Immediately after each fit, left exhaustion hemiplegia is manifest.

Plate IV.

Figure 1.—M. E. H., (Case 15). From a photograph taken three weeks after the series of convulsions illustrated in Figure 2. On the *left side* there is an old infantile hemiplegia which is shown by the flaccid muscles and the bone and muscle atrophy most marked in left arm. The gait of this side is spastic, typical of organic hemiplegia. On the *right side* there is exhaustion-paralysis which has been induced by many hundreds of convulsions. The right arm which was most involved, still shows some rigidity and slight paralysis and contracture. Right hand in athetoid position; the gait on the right side is ataxic but is improving. Recovery, in this case, was complete, the seizures eventually becoming infrequent. It is interesting to contrast the two paralyses illustrated in this case, that of the left side, due to the old infantile hemiplegia (organic) and of the right side which is attributed entirely to exhaustion.

Figure 2.—This photograph shows a general convulsion, except in left hand which remained uninvolved during this series of attacks. The preponderance of convulsion is decidedly on right side (the old hemiplegia being on the left side).

Figure 3.—Photograph of a hand posed in the convulsive position, shows better the position of right hand in Figure 2.

Figure 4.—Also from a pose, illustrates the flaccid condition of the involved left arm and hand in Figure 2.

Plate V.

Figure 1.—L. B., (Case 18). An idiopathic epileptic. Subject to seizures both partial and general which are preceded by an aura in the right arm, or may be induced by irritation of the "Epileptognic Zone," shown in the photograph of the right arm. In many of the partial attacks and some of the grand mal convulsions consciousness is retained.

Figure 2.—C. G. W., (Case 2). Photograph was taken immediately after convulsion of right face (facial spasm) and right half of tongue. The picture shows a slight deflection of tongue to right and obliteration of the naso-labial fold of the same side. This temporary exhaustion-paralysis disappeared in twenty seconds. Speech continued thick and indistinct for three minutes. Consciousness retained throughout.

Figure 3.—W. S., (Case 17). An idiopathic epileptic. In general tetanoid spasm of ten seconds' duration. Convulsions involving the whole body, except the face. The left arm and right leg, which are the parts most involved, subsequently exhibited the phenomenon of exhaustion-paralysis for a few seconds. Similar attacks to this one have been produced by tapping for knee- and wrist-jerks of right knee and left wrist, and are in all respects typically epileptic. In early epileptic career, patient had post-paroxysmal exhaustion paraplegia of lower extremities of two weeks' duration. After grand mal attacks, with left side predominating, he had transitory left exhaustion hemiplegia.

Fig. 3.

Fig. 4.

Fig. 2.

Fig. 2.

Fig. 3.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1.

Fig. 3.

Fig. 4.

Fig. 1.

Fig. 2.

EXHAUSTION PARALYSIS.

Fig. 1.

Fig. 2.

Fig. 3.

II.

PARAMYOCLONUS MULTIPLEX ASSOCIATED WITH EPILEPSY.*

INTRODUCTION.

In this paper our purpose is to present some general introductory remarks on the condition known as paramyoclonus, and particularly to review the development of our knowledge of this condition associated with epilepsy. In addition to a résumé of twenty-seven (to twenty-nine) recorded cases of paramyoclonus associated with epilepsy, which have been reported by Unverricht (two groups of cases), Lundborg, Seppilli, Garnier and Santenoise, Krewer, and Homen, I am able to give in detail the history and manifestations of a case which has been under my own observation, and to present certain conclusions from this case.

Before entering upon the special subject of the paper some brief introductory remarks on paramyoclonus multiplex may be of service to the general reader.

By some writers the identity of this disease is still questioned as it is classed among the protean symptoms of hysteria. Most authorities, however, recognize it as a separate disease belonging to the class of functional neuroses.

This rapid, involuntary, non-systematized contraction of the muscles similar to the muscular response to an electric shock, is closely allied to the choreas on one hand and saltatoric spasm on the other. The affection resembles most, perhaps, Dubini's disease or electric chorea, although the text-books differentiate the latter by various distin-

* Read before the New York Academy of Medicine, Section in Neurology and Psychiatry, April 27, 1899.

guishing features. As only about sixty cases are on record this condition may still be regarded as of rare occurrence. Many of these recorded cases can hardly be called typical, although some degree of latitude must be allowed from the classic type. The affection is usually characterized by sudden, rapid, paroxysmal muscular spasms of a clonic character, involving chiefly the trunk muscles, and less frequently the muscles of the thighs, arms and face. The paroxysms may be of a few seconds or minutes' duration, or may last for hours. The patient may have periods of freedom from paroxysms for days or even weeks. Frequently the paroxysms occur in series lasting for a day or so and then disappear for a period. Thus the patient's life is divided into "good" and "bad" days depending upon the presence or absence of the spasms.

The pathology and pathological anatomy of the disease are quite unknown.

The etiology, beyond emotional or physical shock and the possible presence of neurotic family history, is very indefinite. The affection, likewise, remains in the category of other functional neuroses in point of our knowledge of its real nature. The treatment also is empirical, probably a rational, non-stimulating system of quiet living, preferably in the country, combined with a mild use of sedatives on "bad" days and active moral treatment is as good a general rule to follow as any.

Chapter I.

BRIEF HISTORICAL ACCOUNT OF THE ASSOCIATION OF PARAMYOCLONUS WITH EPILEPSY.

Unverricht seems to have been the pioneer in calling attention to the occurrence of paramyoclonus in epilepsy, or at least the association of the two groups of manifesta-

tions. In 1891 he published his first series of five cases, and in 1895 a second series of three cases. Both sets of cases agree in cardinal characteristics. The cases in each group belonged to the same family—were brothers and sisters—and the myoclonic attacks were preceded by and remained, as a rule, coexistent with epileptic attacks. The myoclonic spasms then occurred in the interparoxysmal epileptic periods.

In his recently published work—Nervenkrankheiten (1898)—Oppenheim says of this clinical condition: "Unverricht has described a particular form of myoclonus which is a family disease and occurs conjoined with epilepsy. In some of his cases the tongue and pharynx are involved in the contractions; also the diaphragm. Weiss, Krewer and Seppilli have described cases." Oppenheim, however, is mistaken in his reference to Weiss, as none of the family reported in this paper* were epileptics, though apparently no less than four generations were involved, and ten separate cases are cited. Lundborg's fourteen cases occurred in the family type of myoclonus. The entire fourteen had myoclonus with typical epilepsy.

Weiss tabulates all reported cases from Friedreich's original observations to date of his own paper (1893). They number fifty-one aside from his own cases. Of these fifty-one only Unverricht's first cases are mentioned as having epilepsy as a complication. Weiss, however, says elsewhere that Ziehen saw two epileptics who had general myoclonic paroxysms between the epileptic attacks. Ziehen reported several cases of paramyoclonus where no mention of epilepsy occurs, and hence evidently did not regard the "general myoclonic paroxysms" seen in the two epileptics as true paramyoclonus.

* Weiss: Ueber Myoclonie (Paramyoclonus Multiplex Freidreich) Weiner Klink., 1893, p. 117.

I have been able to find reports of but twenty-seven cases of paramyoclonus associated with epilepsy, namely: Unverricht, eight cases (family types); Lundborg's fourteen cases (family types); Seppilli, three cases; Krewer, one case; Homen, one case. The two cases of Ziehen cited on the authority of Weiss, must be disregarded as Ziehen seems to have left no record of them.

Chapter II.

UNVERRICHT'S SERIES OF CASES.

In this paper* Unverricht reports four sisters and a brother who were affected in a family of which the parents and a number of other children were healthy. No history of neuropathy or intermarriage. In all the affected children the paramyoclonic symptoms were preceded by epileptic attacks which began in the same manner. Attacks of clonic convulsions occurred nocturnally at an age which varied in each case. These convulsive attacks ran into tetanoid spasms. The urine was voided and consciousness nearly lost. During the first year the attacks occurred about once a month, oftener during second year, and chiefly at night. Whenever consciousness was retained incontinence of urine did not occur. At about the third year of the epileptic seizures, new phenomena, the myoclonic symptoms, appeared, which commenced by muscular contractions of the extremities, during the day. (See Text-Plate A, Figure 1). The nocturnal convulsions became less frequent. The muscular contractions became more general and were distributed gradually over the arms, legs and trunk, becoming also more intense. (See Text-Plate A, Figure 2). Each child

* Not accessible in the original; cited by Krewer.

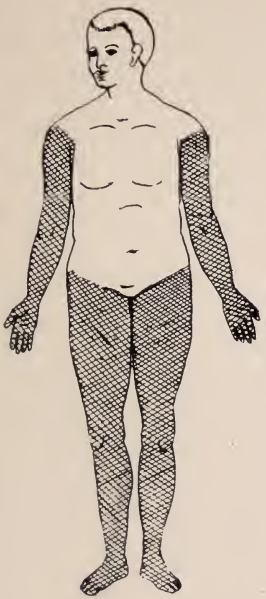


FIG. 1.
Unverricht's 1st Series.

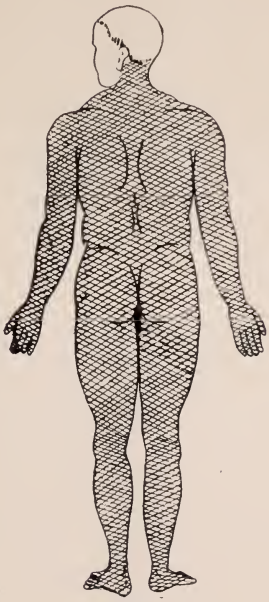


FIG. 2.
Unverricht's 1st Series.

FIG. 3.
Unverricht's 1st Series.

FIG. 4.
Unverricht's 1st Series.

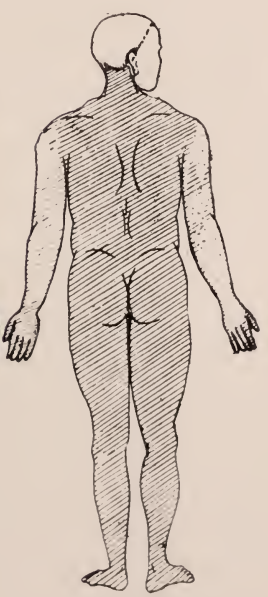


FIG. 5.
Unverricht's 2d Series.
Case I.

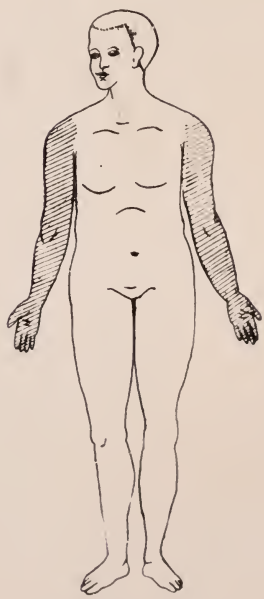


FIG. 6.
Unverricht's 2d Series.
Case I.

//=areas involved in epileptic fits.
::=areas most involved in epileptic fits.
#=areas involved in paramyoclonic spasm.
■=areas most involved in paramyoclonic spasm.

had "good" and "bad" days. Gradually speech participated and the children began to stutter, and several of them also to have difficulty in swallowing. All the children had been taught reading, writing and arithmetic and showed proper mental development. Nervousness, headache, etc., were not present. The symptoms were refractory to all treatment.

These five cases resembled one another so strongly that in citing Krewer's details of one case it practically holds true of the whole group. An abstract of the cases is as follows:

There are lightning clonic movements of the muscles which occur at irregular intervals and involve one or both of the upper and lower extremities and usually the trunk. The following muscles are involved, mostly on left side, as shown in Text-Plate A, Figures 3 and 4: Sterno-mastoid, pectoralis major, deltoid, biceps, coraco-brachialis, supinator longus, extensores carpi et digitorum, trapezius, latiss. dorsi, serratus magnus, teres major et minor, erector spinæ, extensores (mostly) flexores et adductores femoris, tibialis anticus, extensores pedis and the calf muscles. The tongue when protruded twitches throughout and the borders are scarred from bites. The even flow of speech is interrupted. As a rule symmetrical contractions are absent; only rarely does it happen that the corresponding muscles of both sides move together. Oftentimes one extremity shows stronger contractions than the other. It not infrequently happens that there is a pronounced muscle play without any corresponding locomotive effect, especially in the extensors of the forearm and hand.

Unpleasant emotions appear to cause an increase in the contractions. No impediment to the seizing of objects but if anything is held in the hand at arm's length, contractions occur.

Walking occasions much trouble at the start. As soon

as the patient gets up from the sitting posture and while the first steps are being taken violent movements of the whole body set in, especially in the legs. A series of steps can then be taken if patient is supported by some one. The special senses and reflexes show nothing very unusual. Indican is present in the urine.

The four other affected children show hardly any departure from the foregoing description. The youngest was kept apart from the others during a period of six years before her attack, yet she developed the disease like the others.

Weiss also gives a short abstract of these five cases of Unverricht's, and says that "the contractions stand in a causal connection with the epilepsy." The latter began with nocturnal attacks when the children were from six to thirteen years of age. These attacks were often preceded by an aura.

Later the epileptic attacks became weaker and more infrequent, but there set in diurnal attacks of paroxysmal clonic contractions, in which all but the eye muscles took part. On "good" days the face was free, on "bad" days and usually before epileptic attacks the face also participated. In the interval between paroxysms ordinary movements, such as writing, could be performed. The close connection between these spasms and epilepsy is unmistakable.

We find that the cases in Unverricht's first paper were not quite classic. The extremities were involved below knee and elbow occasionally, and at times the facial muscles were involved.

Reynolds claims that 75 per cent of epileptics have tendency to more or less abnormal movements varying between paroxysmo-tremor and tonic and clonic contractions.

In my experience, Reynolds' claims are altogether too high. I do not believe that more than 10 per cent of all epileptics have disorders of motion in the interparoxysmal state. Certainly not more than ten or twelve in over five hundred cases which I have personally examined in the past four years, showed abnormal movements of a spasmodic nature as a consequence of their epilepsy. This statement justly excludes disorders of motility in hemiplegic epileptics where the disturbance is just as independent of the epileptic phenomena as the atrophy and vaso-motor disturbances are. Many observers admit the comparative frequency of "myoclonic spasm in the interparoxysmal state" noted in Ziehen's two references.

Chapter III.

UNVERRICHT'S SECOND SERIES OF THREE CASES.

These three cases in a general way resemble those described in Unverricht's first paper. All were children of the same family, and both the epileptic and myoclonic symptoms appeared before the age of twenty.

In the first case a trauma was succeeded by two attacks in the course of several weeks and during this time the myoclonic contractions appeared. The contractions and epileptic attacks continued for eight or nine years, the latter occurring about twice a month. In the second case the epileptic attack and myoclonic spasms apparently came on simultaneously. The epileptic attacks persisted with the myotonic spasms. In the third case the myotonic contractions were ushered in by a few attacks of epilepsy which ceased spontaneously.

Case 1.—Male, age 32. Father died of phthisis; mother is living and well; a brother aged 28 is healthy; a sister died of phthisis at 16; two other brothers, aged 20 and 17,

have the same disease as this patient. The patient had always been well until at the age of 15 he was thrown from a horse, receiving a head injury which confined him to his bed for a fortnight. During this time had two epileptic attacks with opisthotonus, flexion of the arms and loss of consciousness. The tongue was bitten in second attack. The areas involved in epileptic convulsions are indicated in Text-Plate A, Figures 5 and 6.

Several weeks later, weather being cool and moist, contractions began in the cervical muscles, principally of the right side, the head being drawn backwards. About same time contractions of arms, legs and body developed. Since this time patient has never been free from contractions except for a few moments at a time. Their intensity varies considerably, and they do not cease entirely during sleep. For eight or nine years he has been unable to work. As a boy he was punished for his contractions.

About twice a month, on an average, patient has an epileptic attack with loss of consciousness and the tongue is bitten. When the attack does not result in quieting the contractions it is because a second attack is impending, after which he becomes easier.

Cold, fright, vexation and moist weather increase the contractions. All voluntary efforts to suppress the contractions have a contrary effect. He often falls to the floor, drops things he is carrying, and even wounds himself with knives.

The Present Condition (recorded after several weeks of observation) is as follows: Patient is healthy, well developed, head of normal shape, senses normal. V=½. Derangement of color perception. No nystagmus nor contraction of eye-muscles. Contractions elicited from jaw-muscles (masseter, temporal). As patient sits quietly facial muscles are at rest. If ordered to make grimaces the muscles concerned are affected by lightning-like contractions. Symmetrical muscles are not affected. The orbicularis palpebrarum, levator anguli oris and corrugator supercilii actively participate. On "good" days there are no disturbances; only at times speech is inter-

rupted in the middle of a word through muscular contractions. There are days in which the contractions are markedly increased. On very "bad" days conversation is interrupted by long pauses. The tongue has no prominent scars, is protruded promptly but shows fibrillary twitching. Occasionally it is protruded or drawn in with lightning-like rapidity. Swallowing is normal. Gait shows nothing abnormal as a rule. When patient is aware that he is being observed or believes that he is about to have a fit, he is unable to go about because of the violence of his contractions in the most unused muscles. He may be thrown to the floor, his knees may give way, or he may stamp his feet or stumble. Contractions affect single muscles rather than groups. These single muscles do not have isolated innervation. At times only portions of a muscle contract, *e. g.*, the pectoralis major. The corresponding muscles of both sides are not synchronously affected, *e. g.*, the left platysma is usually quiet while the right is very often affected. Predominance of either side of the body in the spasm is not evident. Nearly all muscles participate excepting those of the eye. The muscles of neck and trunk are most concerned, as shown in front and back views in Text-Plate B, Figures 7 and 8. Voluntary motion does not affect the contractions. It neither strengthens nor checks them. The locomotive effect is marked. The body is flexed or extended, head drawn backwards or to one side. Emotional states increase contractions. If the patient is surprised while about to perform some voluntary act there is a lively muscle play. During sleep contractions do not cease but the frequency and intensity are diminished. General examination of senses, reflexes, etc., reveals nothing of importance. Indican occurs in the urine.

Case 2.—Male, age 20; brother of Cases 1 and 3. Always well up to the thirteenth year, when contractions and convulsions set in, the former appearing "in the spring of 1883" while the latter first occurred "in May, 1883."

The *first attack of epilepsy* began in the right orbicularis palpebrarum with the face turned to the right shoulder, with spasms first of the upper, then of the lower extremities, worse on the right side, as shown in Text-Plate B, Figure 10. Patient was thrown to the floor and lost consciousness, awakening later from a deep sleep. Two weeks later had a similar attack and from then on the attacks occurred regularly every week or ten days.

The epileptic attacks usually began in the early morning hours. He would first be awakened and then lose consciousness. The urine is usually voided during paroxysms. After the first seizure (which began in the orbicularis palpebrarum), the attacks began with a flexion and pronation of the right great toe. An attack has never started from the left side of the body. The tongue is often bitten. After an attack the contractions are less marked.

The *contractions* began first in right half of face and tongue, and right side of orbicularis oris. The left side was soon afterwards affected similarly but less severely. There was occasional hiccough. A few months later contractions of right arm and soon after in the left arm but weaker. Next in order came the right followed by the left leg, the left side being invariably weaker. At about the same time the trunk was affected. Both sides are not synchronously affected. For areas involved in the paramyoclonic contraction, see Text-Plate B, Figure 9.

The contractions differed in intensity. There were "good" and "bad" days, although the patient was never wholly free. The tendency was always for the worse, so that he was finally incapacitated from work. They become more violent twenty-four hours before a paroxysm or after having worked hard. When sitting in repose, there was only a slight muscular impulse. Motion, cold and the unpleasant emotions all aggravate attacks. After a Turkish bath the contractions are much less marked and the patient feels well. He also feels much better in summer than winter, and in pleasant than in bad weather. During sleep only single and weak contractions occur, but when he turns in bed they become so violent that they

awaken him. After the first epileptic attack the contractions became more violent, enough so to throw patient suddenly to the ground.

Present Condition.—Patient is well built. Head well formed. No tenderness of head and spine on percussion. The senses and reflexes, show nothing peculiar. The gait is normal; on "good" days he can even run. As patient sits quietly, twitchings of the orbicularis palpebrarum are visible, but these are not synchronous on both sides; the right side preponderates as shown in Text-Plate B, Figure 11. Contractions of sterno-mastoid, also worse on right side. Facial muscles are occasionally involved but there is never any twitching of the muscles of the bulbi oculi.

When the mouth is open the tongue is seldom quiet. It is thrust out, drawn in and moved laterally. When asked to protrude the tongue, the patient cannot at first respond as strong contractions take place, and when protruded it cannot be kept still. Swallowing is not affected.

Case 3.—Male, age 17; has had no illness except measles at 6, smallpox at 14 years. Like his brothers he had been well brought up. When about 15 years old was found one morning unconscious; tongue had not been bitten; felt well on recovery. One month later he was suddenly taken with muscular contractions in the right arm, these were not however marked enough to interfere with his work. A few days later the same thing happened to the left arm, the eyes, the facial muscles and finally the lower extremities.

Six weeks after the first seizure, just as he was rising from bed, he experienced a second convulsive attack in which he suddenly lost consciousness and fell back in bed. The body was shaken with spasms, there was frothing at the mouth and bladder was emptied. He waked later with a bitten tongue and heavy feeling in the head. *Since that time has had no further attack of epilepsy.*

The muscular contractions were originally absent for days at a time, but latterly they have become more fre-

quent and violent. Contractions are especially marked after corporeal efforts, cold, fear, vexation, etc. Patient believes that when his attention is not relaxed, he can limit the intensity of the contractions but cannot wholly suppress them.

Up to three months ago, patient was free from contractions during sleep; since then they have appeared during sleep but in a mild degree. He notices them especially when he turns in his sleep. When he becomes tired his writing shows the effect of the contractions.

An examination of his present condition is chiefly negative. There is no indican in urine. By watching the patient awhile, one perceives lightning-like muscle plays throughout the body. Single muscles alone are affected. The movements called forth are not enough to interfere with patient's work. The facial muscles contract visibly. If asked to close his eyes a continuous tartiling of the orbicularis results which is not perceived at all when the eyes are opened. When protruded the tongue shows fibrillary twitchings which extend uniformly on both sides. There are also twitchings of the orbicularis oris. Active and passive mobility, as well as the general energy, is fully preserved in all the limbs. When patient awakes in the morning the contractions which have been quiet during the night begin afresh and are then confined to the facial muscles. Only a part of the pectoralis major contracts at times. The paroxysms were also general at times, even involving the distal portion of the upper and lower extremities and muscles about eyes and mouth.

Again, we see that Unverricht's second series would not be considered classic by those who hold that the muscular contractions must be distinctly paroxysmal, leaving the patient entirely free in the intervals.

FIG. 7.
Unverricht's 2d Series.
Case I.

FIG. 8.
Unverricht's 2d Series.
Case I.

FIG. 9.
Unverricht's 2d Series.
Case II.

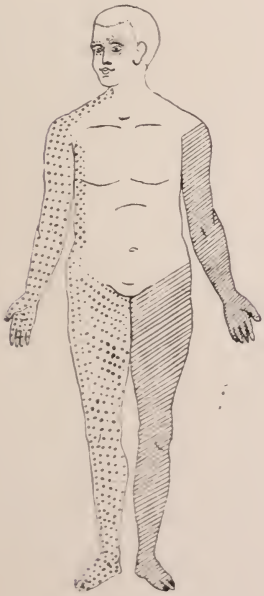


FIG. 10.
Unverricht's 2d Series.
Case II.

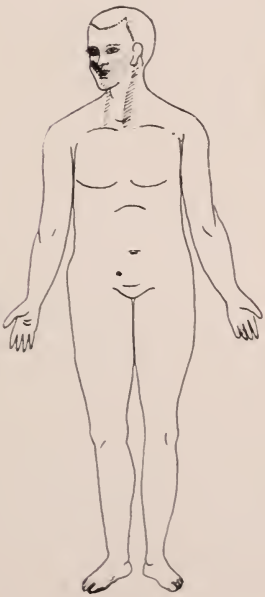


FIG. 11.
Unverricht's 2d Series.
Case II.

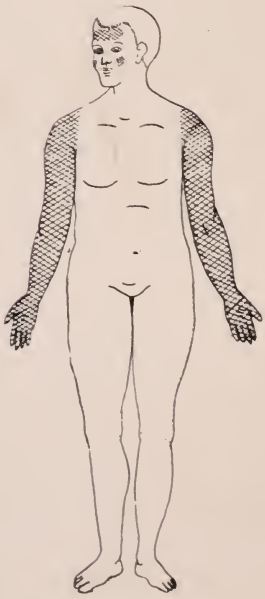


FIG. 12.
Seppilli's Cases.
Case I.

//=areas involved in epileptic fits.
::=areas most involved in epileptic fits.
#=areas involved in paramyoclonic spasm.
■=areas most involved in paramyoclonic spasm.

Chapter IV.

LUNDBORG'S CASES.

Since the collection of the data for this paper Lundborg has reported in full a most remarkable instance of family myoclonus in which fourteen cases of *associated* myoclonus and epilepsy are presented in various members of seven families of one stock. It would seem that he strongly believes in the production of paramyoclonus, with or without other attendant degenerative neuroses by the intermarriages of first cousins who are neuropathic.

In considering Lundborg's paper, we will give in abstract, first, a sketch of the entire stock which produced the fourteen cases of myoclonus and epilepsy; second, a few notes of the seven distinct families from which the cases were derived; third, a short résumé of the individual patients; fourth, the general consideration of the disease as conceived by Lundborg (etiology, course, prognosis, etc.) Lundborg was apparently unaware of the publication of a number of other cases, and his paper is chiefly valuable for the report of his own material.

He relates the history of several generations of an intensely neuropathic stock, which exhibited, among other neuroses, the association of paramyoclonus with epilepsy (fourteen individuals representing seven families, being thus afflicted). Three elaborate genealogical tables are appended, indicating both the relationship and the neurotic pecularities of the various individuals.

The following neuroses are especially prominent. Tic convulsive, paralysis agitans, ordinary epilepsy, and epilepsy associated with myoclonus. Other non-neurotic conditions, such as obesity, tuberculosis, etc., are also noted. The ancestor of this family was Per P., who was

born 1720, and died 1803. The second generation comprised three children, Bengt, Per, and Elsa. The descendants of each of the above showed the same neuropathic taint.

Descendants of Bengt.

(Second Generation).

Only one child was recorded, a son, in the third generation. This son had four children (fourth generation), and here we find the first indication of psychopathy, the second child being a suicide. The members of the fourth generation (Per N., Knut and Sven) left issue. In the fifth generation Knut the son of Per, committed suicide like his uncle. Several consanguineous marriages are noted in the fifth generation. In the sixth generation, we note among the few descendants of Per (of the fourth), an idiot and an epileptic (this line of descent appears to have become extinct). It should be stated that the two suicides both suffered from tic convulsive, as did also Sven (of the fourth). The suicide, N. P. in the fourth generation, had a daughter with tic convulsif, and three of the children of Sven (of the fourth), also had the same malady, this making seven cases of tic, in the descendants of Bengt (second) as far as the fifth generation.

We are now down to the sixth generation, where we find one idiot, and one epileptic, in the family of N. P. (of the fifth); two ordinary epileptics and three epileptics with myoclonus in the family of Nels (of the fifth,) who had married his cousin; and one epileptic with myoclonus in the family of Knut (of the fifth).

We have here documentary evidence that a marriage of first cousins of a tainted stock, produced both epilepsy, and epilepsy plus myoclonus. The latter affection does not appear to have occurred alone.

Descendants of Per.
(Second Generation).

The third generation of this branch was represented by three children, Sven, Elsa and N. and no ailments are recorded concerning any. But in the fourth generation, in the children of Sven and N., both of whom married cousins, we find epilepsy with myoclonus. Of the four children of Sven, two were epileptics with myoclonus, and a third had paralysis agitans. The fourth child, a female, married a cousin, and the issue was another epileptic with myoclonus. The children of Elsa (of the third), had three children. Two of these were seemingly healthy, but the third had paralysis agitans. N. P. (third generation), who married his cousin, had six children (fourth generation), and of these, two had epilepsy with myoclonus. With regard to the fifth generation, we find one epileptic with myoclonus, one plain epileptic, and three cases of paralysis agitans. Finally, in the sixth generation, in which the stock is largely extinct, we find one case of simple epilepsy; a case of tic is found in a member of the fifth generation.

Descendants of Elsa.
(Second Generation).

The third generation is represented by two healthy children, Nil (of the third), had Bengt and Nils (fourth generation), and each married a cousin, daughters of Anna (of the third). Of the fourth generation we find two epileptics with myoclonus, children of Anna (of the third). The descendants of this Anna are not carried further (probably becoming extinct). In the fifth generation we find three epileptics with myoclonus, and three cases of tic in idiots and imbeciles. Only one member of the sixth

generation is given, and he is an ordinary epileptic. Here, as before, we find the epileptic myoclonus in the offspring of intermarriages between cousins.

Résumé.

Taking this entire stock together, we find the following:

1st generation	(Per P.) healthy?	
2nd "	" ?	
3rd "	" ?	
4th generation	Paramyoclonus and epilepsy	6
	Tic convulsive	2
	Paralysis agitans	2
5th generation	Paramyoclonus and epilepsy	4
	Plain epilepsy	1
	Tic convulsive	9
	Paralysis agitans	3
6th generation	Paramyoclonus and epilepsy	4
	Plain epilepsy	5
	Tic convulsive	0
	Paralysis agitans	0

Numerous other neurotic peculiarities are incidentally mentioned, idiocy, imbecility, paralytic dementia, suicide, etc.

Individual Families.

From the general review of the stock as a whole, a study of those families which especially exhibit paramyoclonus plus epilepsy is of interest.

Family I.—Knut P. married his kinswoman; his son Nils married his cousin—three children had paramyoclonous with epilepsy, while two others had ordinary epilepsy.

Family II.—Sven P. married his cousin; two children had paramyoclonus with epilepsy; a third married her own cousin Sven S., and had a daughter with the same association of disease.

Family III.—The results of the marriage of Sven S. are given under Family II.

Family IV.—N. P. married his cousin and had two childred who had epilepsy with paramyoclonus.

Family V.—Bengt N. married his cousin and had one child with epilepsy plus paramyoclonus.

Family VI.—Nils N. married his cousin and had two children with paramyoclonus and epilepsy.

Family VII.—Anna N. married a man to whom she was *not* related: They had two children who had paramyoclonus plus epilepsy.

Thus of these seven families all but one marriage was of first cousins.

Individual Cases.

A brief synopsis of each case of paramyoclonus and epilepsy is given.

Case 1.—Female, age 26. Symptoms began at the age of nine or ten years of age; paramyoclonus appeared first, and was soon followed by epilepsy. Patient is an imbecile. Disposition obstinate in the greatest degree. Swallowing is difficult, and on certain days almost impossible. Clonic movements are increased by the emotions, embarrassment, fright, anger, etc., and at the time of menstruation; they are diminished by sleep, by diverting attention, and by fever and the action of coffee. Patellar reflex is marked. In this case the contractions were uniform in strength and frequency. Boulimia is present. There are present, at times strong hyperidrosis and polydipsia.

Case 2.—(Sister of Case 1). Female, age 22. An imbecile and has pulmonary tuberculosis. Movements began at the age of nine or ten years of age. Disposition is violent. There is no further data, save that the attacks exactly resemble those of her sisters.

Case 3.—(Sister of the two preceding). Female, age 20. An imbecile; she has pulmonary tuberculosis and chronic heart disease. She is refractory. The paroxysms

resemble in every way those of her two sisters; they began at the age of ten years.

Case 4.—Female, age 62. Has obesity and progressive dementia. Her intelligence had been good. Disposition is violent. Her movements are diminished by chloral hydrate and alcohol, and by pregnancy. She is especially erotic.

Case 5.—(Sister of Case 4). Female, age 40. In both her and her sister, symptoms first appeared at the age of nine years. Obesity is present. Movements are diminished by sleep, chloral, and alcohol. Her disposition is violent. Markedly erotic.

Case 6.—Female, age 39. Attacks began at the age of ten years. She is imbecile and alcoholic. Swallowing is difficult at times. The movements are lessened by sleep, fever, alcohol, bromides, and pregnancy. There is marked hyperidrosis and polydipsia on certain days.

Case 7.—Female, age 49. Her attacks began at the age of 10 to 14 years of age. Has obesity and organic heart disease. Her intelligence is good. The movements are diminished by sleep and coffee.

Case 8.—(Brother to Case 7). Male, age 55 years. Attacks set in when 13 years of age. Has obesity and progressive dementia. The movements are lessened by sleep and alcohol.

Case 9.—Female, age 34. Her symptoms began at the age of ten years. Epilepsy appeared only after the lapse of many years. She has good intelligence. Her disposition is violent. The movements are increased by psychical factors and menstruation, etc., and are diminished during sleep and fever. Boulimia is present. Marked hyperidrosis and polydipsia are present.

Case 12.—(Brother of Case 9). Male, age 46. The symptoms began at the age of 30 years. The movements are diminished by sleep and alcohol.

Cases 13 and 14 are not considered in detail.

Lundborg's inference upon the etiology of the cases in his study, is that the female sex appears to predominate markedly over the males, the proportion being two or three to one. The age of nine or ten, or the interval between the ages of seven and fifteen is by far the most frequent period for the disease to begin. Heredity of course, plays a marked rôle, not direct inheritance, but, neuroses, alcoholism, etc. There is no example of a paramyoclonic epileptic begetting a similarly afflicted child; and there are a few cases in which a general neuropathic inheritance is absent. Many of the progeny of the patient enjoyed apparent health but all died of some intercurrent somatic affection (tubercular meningitis, etc.)

In considering the relative time of development of paramyoclonus and epilepsy Lundborg believes from his experience, that in some cases epilepsy develops before myoclonus, while in other instances the myoclonic movements precede the epileptic convulsions, by one, two, or three years, particularly in those cases where the myoclonic movements develop between the eleventh and twentieth year.

The coexistence of heart disease or of the tuberculosis with myoclonus which occurs in several cases, is considered by Lundborg as a coincidence.

One of Lundborg's cases which had had the myoclonic movements for thirty years, had during that time, but two epileptic seizures.

In regard to the development and course of disease and prognosis, Lundborg calls attention to the fact that most of his cases have died during the observation period. He makes three stages of the affection.

First, initial stage, which begins with the first appearance of either epileptic or myoclonic symptoms, and

extends to the period at which both symptoms coincide. This period is usually coincident with puberty.

Second stage, which extends to the maximum development of the symptoms, usually the forty-fifth or fiftieth year, he terms the "climacteric stage." Its close is marked by various degenerative and permanent alterations both psychical and physical. The death of those in the second stage were due to intermittent diseases, chiefly tuberculosis and heart disease.

Third, or post-climacteric stage, extends from the age of fifty or thereabout to the time of death. But four of Lundborg's cases reached this third stage, and of these, three died of progressive dementia.

Lundborg concludes that myoclonic movements, with a tendency to periodicity and developing in an epileptic, are evidence of so-called family disease, and are destined to continue throughout the patient's life.

It is of signal moment to notice that we find included in this paper two distinct types of cases—family myoclonus plus epilepsy, and sporadic cases of the same combination. Of the family type, Unverricht's and Lundborg's cases are typical; and of the sporadic type, Krewer's and the author's case are illustrative.

Attention is called again to the fact that in the association of the two diseases the classic character of each suffers more or less; but there can be no doubt, in the mind of one carefully weighing the evidence that the association of the two diseases—paramyoclonus and epilepsy—in the same case, marks that individual as typically degenerate.

Chapter V.

SEPPILLI'S THREE CASES.

These three cases form a group with the salient feature of the two sets of manifestations occurring in two brothers and a sister of the same family. The age when the fits and spasms began is also striking as it occurs in adolescence as in the cases of Unverricht. In the first case epileptic fits precede the myotonic spasms by a space of three years.

In the other two cases the epileptic attacks make their appearance first, although they continue along with the myotonic symptoms.

The family history contains nothing of significance beyond the fact that the father was an alcoholic and had been imprisoned for an assault. He had abused and neglected his children as well as their mother during her pregnancies. The mother was healthy and sane. She had eleven confinements, three children dying in infancy. Of the survivors, all were perfectly well excepting the three with paramyoclonus.

Case I.—Male, aged 22; three years before while in prison for theft, epileptic attacks began. He was violent and quarrelsome by nature and after an attempted assault upon a sister was removed to the asylum.

He is short, slender, somewhat effeminate in appearance, the head well shaped, ears and zygomata projecting and prominent. Nothing abnormal in the senses and reflexes. The gait is regular, free and even, save that now and then an abrupt contraction occurs in the leg of one side or the other, compelling him to carry his foot outward and to plant it forcibly on the pavement. Sometimes the contractions are so intense that they upset the equilibrium of the body and patient is liable to fall. This occurs

especially on slight rotation of the body, consequent to changing the direction in which he is walking.

The standing posture is well maintained for a time. Then the violent tremor attacks the legs and is diffused to the abductors of the arms and hands (rapid contractions of the extensor and flexor communis and extensors of the thumb); the facial muscles are also affected (clonic contractions of zygomatic and frontal muscles). (See Text-Plate B, Figure 12). Patient can execute some voluntary movements, but as soon as a voluntary act is complete or even during perfect repose, the brusque muscular contractions set in and derange the limbs. There are muscular oscillations of certain muscle groups (flexors or extensors of hands or fingers, the sartorius and the tibialis anticus) of one or both sides of the body, or clonic contractions of both sides of the face and the back (trapezius, extensors of vertebral column), as seen in the front and back view of Text-Plate C, Figures 13 and 14.

This spasmodic state of the muscles is paroxysmal. On certain days it is absent. On others it is constant, but varying in intensity and diffusion. Ordinarily it exacerbates with voluntary motion and under the influence of the emotions and sometimes both precedes and follows the epileptic attacks to which patient is subject. They are absent during sleep.

Patient learned to write as a boy, but now he takes a long time to write a few words. He grasps the pen with force and presses it firmly against the paper. The letters are a little unequal in size, and at times the writing shows the effect of tremor and spasm.

Articulation of words is clear, the syllables are finished slowly. The words are scanned, and not rarely speech is arrested in the midst of a syllable by a spasm of the lingual muscles. He pronounces forcibly. While he speaks, the facial and frontal muscles contract. These anomalies of writing and speech are especially marked during a paroxysm. Ideation, normal. Memory, good. Disposition, irritable, exaggerated emotions. Moral sense, obtuse.

Epileptic attacks may be followed by a period of mental confusion.

Case II.—Male, aged 29. First epileptic attack at 19. Was qualified for military service, but after six months was weeded out because of his fits. After his return home began to have tremor and muscular jerks which became notably worse. Was violent, aggressive, and addicted to alcohol and was finally placed under restraint.

The patient is robust and well-developed with projecting ears and prominent zygomata and large occiput. The senses and reflexes are normal.

At longer or shorter intervals a muscular shock in the arms and tremor in the legs occur. Extension of the hands with contemporaneous spreading of the fingers accompanied by slight tremor of the latter. The hand is readily carried to the head, mustache curled, glass raised to lips and all other complex movements are finished normally. Sometimes a voluntary movement is interrupted by a brief muscular contraction, and followed in a few seconds by another. Standing erect on one leg gives rise after a time to a tremor of the limb which is the more intense the longer the patient holds himself in that position. In horizontal decubitus during repose there are fibrillary oscillations of the muscles, in one or another group, often bilateral. On some days the gait is normal. On others it is associated with muscular "shocks" of the legs which occur instantaneously and violently at long intervals and "shock" the entire body.

In speech the syllables are formed regularly but follow one another slowly, with pauses. Sometimes the sounds are explosive. Facial and frontal muscles contract during speech. Writing is slow, tremulous, and at times zigzag, due to muscular contractions of the arms. Contractions cease during sleep and are more marked before and after the epileptic attacks. The latter are classical in character.

Case III.—Female, age 31. Epileptic attacks began at 11 years of age, occurring chiefly at night. Three years later spasmodic tremor began, which was worse

after the epileptic attacks. Was admitted to the asylum for persistent agitation. There are muscular contractions in limbs while standing or walking. In horizontal decubitus during repose there are fibrillary twitchings in certain groups of muscles in the extremities, or brusque contractions like an electric shock. Muscles of face, neck and trunk also subject to spasmodic contractions. Emotional state causes exacerbations. Voluntary acts are normally completed excepting when interrupted by muscular contractions. Speech and writing are like the brother's. The spasmodic state is a constant one but varies in intensity and diffusion. Especially marked before and after epileptic attacks.

In Seppilli's cases we find the paramyoclonus occurring after as well as before the epileptic fits. This is very exceptional. I have noticed it only two or three times in my case. In epilepsy it is not uncommon to have those same motor and sensory phenomena which preceded the fit follow the seizures; so this fact does not materially militate against the possibility that the association of the two diseases is dependent on the same pathological conditions.

Chapter VI.

KREWER'S CASE.

In Krewer's case the myoclonic contractions also occur in a juvenile patient. Epileptic attacks began at the age of thirteen and after five years became associated with the myoclonic spasms, which at the beginning were weak and feeble but gradually increased in severity. The myoclonic spasms in this case seemed to have a distinct association with the fits, for when the spasms increased in severity the epileptic attacks became more frequent, occurring every day. Probably the close relation of the

myoclonic phenomena to epileptic manifestations is best shown in this case and my own. The details of the case are as follows:

Female, age 19. Parents always healthy. Mother was twice married. By first marriage there were four children, all of whom died in infancy; by the second marriage there were two children, the patient and a brother, age 30, who is healthy. Excepting the myoclonic condition the patient was perfectly healthy until age of 13. She is well-built, strong and not anæmic. Hands showed many scars from self-inflicted wounds during the paroxysms of contractions. Special senses and reflexes presented no great deviation from the normal. The orbicularis oris and levator anguli oris twitch constantly. Other facial muscles contract only in the general paroxysms.

At the age of 13 the first attacks of epilepsy occurred. Consciousness was wholly lost. After an interim of perfect health for three months the second attack occurred. Other attacks followed at irregular intervals for a period of five years or more and during this time the myoclonus had developed as a complication. This set in two years before patient was first seen, and about four years or more after the first epileptic attack. The earliest movements were weak and infrequent and hardly interfered with the patient's needlework. Gradually they became more frequent and violent. There were "good" and "bad" days. On the former, patient could attend to her duties, such as writing and knitting. On "bad" days she could not even walk or feed herself and had to lie abed. Whenever the movements increased in violence the epileptic attacks were more frequent, so that hardly a day passed without a fit, chiefly at night. Attacks were made worse by emotion. Contractions began first in the arms and gradually extended to the legs, trunk and face. Within a year and a half speech had become affected and at its worst was almost suppressed.

The patient admitted October 4, 1895. After one month of observation her general examination was taken with the following results:

Whenever the patient knows she is being observed the contractions are more violent. Contractions are of the lightning order and seldom lead to locomotive results. Nearly the entire musculature of the part affected seems to contract within an instant of time. There is no system, rhythm or regularity; no choice between the two sides. Tendons of forearm and hand twitch violently without any movement of the fingers. At times locomotive effects are observed. These are without purpose, inco-ordinated, sometimes causing flexion or rotation. The limb is hurled, as it were.

The following muscles take part: Deltoid, biceps, triceps, brachialis internus, supinator longus, flexors et extensores carpi et digitorum, vastus externus, semitendinosus and semi-membranosus, gracilis, extensores pedis; more rarely the trapezius, pectoralis major, adductores femoris, orbicularis oris, levator anguli oris. In fact nearly all voluntary muscles of the anterior part of the body are affected as shown in Text-Plate C, Figure 15. The following move only in general paroxysms: Muscles of neck and throat, long back muscles, rectus abdominis and diaphragm, as indicated in black in Text-Plate C, Figures 15 and 16. The muscles are seldom affected in definite groups, contractions being confined as a rule to single muscles or parts of muscles. It might happen, for example, that the right deltoid, left vastus externus and left supinator longus would contract simultaneously. When several muscles in a group do act synchronously we, of course, get locomotive effects. At these times by exertion of will the spasms may be controlled and when they cease the purposive movements are continued. Writing is interrupted and performed in a similar manner.

The frequency of spasms varies. On "bad" days the patient is seldom free, the contractions may even follow one another so rapidly that a brief tonic spasm results (1–2 seconds). These tonic spasms are observed shortly before epileptic attacks, a point of considerable value in prognosis.

Active voluntary movements increase the contractions, not only of the muscles toward which the volition is directed but of all others. Contraction of the former do not occur until the voluntary movements have been executed. The impression made is that the contractions might be checked by the will. The active movements are normal as to co-ordination, but are sometimes hindered by the involuntary action of other muscles. While lying quietly, movements are less active, and during slumber, they almost disappear.

"Bad" days precede and "good" days follow the epileptic attacks, which almost invariably begin at night and during sleep, or rarely, early in the morning, still more rarely in the daytime. They are classically epileptic, lasting two or three minutes, and followed by prolonged and deep sleep. The tongue is seldom bitten, but the urine is usually voided. Patient awakes in good condition unless a second attack is impending. Chloralhydrate was of considerable service in preventing epileptic attacks. Speech is normal only when contractions are wholly absent. Ordinarily, the even flow of speech is broken, interruption occurring in the middle of words, producing an effect similar to stuttering. The disturbance of speech is best explained by the lightning-like character of the movements producing severe shock. The tongue can be protruded straight. Swallowing is normal. Active and passive movements of head and joints are normal. Both sides of body are symmetrically developed. Electrical reactions and muscular sense normal. The gait is irregular, subject to the involuntary action of the muscles. Patient often falls. When the contractions are weak she can walk, but this is unusual. Sometimes the spasm of the diaphragm is enough to interrupt the speech. When the patient starts to get up in the morning the contractions set in strongly, but by a strong effort of will they can be partially suppressed for a moment only. She then hastens to get out of bed. Before she can take a step the contractions are again upon her to a marked degree. She stands by the bed until she can again summon her will-power, and then

walks with quick, short steps until the spasms reappear. She then raises herself on tiptoe, spreads the legs, bends forward and turns to one side. A general series of contractions then follows. In a series of twenty-five tests Krewer found no indican in the urine.

Chapter VII.

HOMEN'S CASE.

Homen, a Finnish neurologist of Helsingfors, describes a case of paramyoclonus multiplex in an epileptic. The association of epilepsy in this case which Homen mentions in a very cursory manner has been overlooked by every one except Unverricht, who cites it in his first paper. Nagl in describing an anomalous case of continuous muscular movements in an epileptic, (*Weiner med. Zeitung*, 1895), also cites this case of Homen's. Nagl's case I reject as not being paramyoclonus, but probably a slight general chorea.

Case I.—Male, age 45, farmer. Seen November 18, 1886. Father alcoholic; mother well. No other children. Always well in childhood. First epileptic fit when sixteen years of age. Soon after this involuntary contractions began in the limbs and face (the latter rare). These contractions produced isolated locomotive effects and occurred in series. They were marked in repose, augmented with motion, hard labor, fatigue and mental emotions. Whenever he drank too much he had attacks of general convulsions followed next day by great prostration and increase in the contractions. During an attack of typhoid fever, the contractions persisted. The man was well developed throughout. His senses, sensation, reflexes, muscular strength, electrical reactions, were all normal and mentality good. Movements of tongue normal.

Contractions of the face were marked and were increased by speaking, the buccal muscles especially, and the

zygomatici most of all. In the arm the whole musculature was not affected at once, the muscles twitching in succession rather than synchronously. The supinator longus, biceps, deltoid, triceps and extensors of wrist were chiefly affected. (See Text-Plate C, Figure 17). The resulting locomotive movements were enough to interfere with handling objects. From twenty to twenty-five contractions would occur in fifteen seconds. As a rule patient wrote well, occasionally a contraction would interrupt. He was not much hindered in his daily work. In the legs the quadriceps, more rarely the semitendonosus, semimembranosus and gluteus major, and still more rarely the adductor magnus, were affected. When he was falling asleep the contractions became worse, but after sleep had supervened they were more quiet. While sitting up in bed he has violent contractions of the back muscles; if he goes to rise, he has lightning contractions in the quadriceps, one hundred or more a minute, with corresponding locomotive effect. These contractions appear to travel upwards along the spine so that when walking without his cane he is in some danger of falling, at times he cannot walk at all.

When he was about to be shown before the Finnish Medical Society he felt a repugnance against being put on exhibition and his contractions became very violent. The movements of flexion at the knee became so marked that he was unable to walk and was carried in on a stretcher, the movements persisting. Two glasses of brandy were given him with the remarkable effect that he got up from the stretcher and walked about.

GARNIER AND SANTENOISE'S CASE.

This case presents nothing particularly new, and seems to be fairly typical of the associated diseases in a degenerate family, like those of Unverricht and Lundborg. The case in the abstract is as follows:

L., a male, age 26, single. He was first seen in 1877,

and had been ailing three years. Parents were living and well. The mother's head suggested degeneracy. A paternal aunt was an epileptic. A younger brother is somewhat backward, and a younger sister has marks of degeneracy.

L. became an epileptic when twenty-three years old, after a fall, resulting in great fright and injury to his leg. Shortly after the wound healed (nine days after injury) the first spasms occurred. These first spasms were of the paramyoclonic type and in no sense epileptic. They were limited to the eyelids and the upper extremities, and were present during meals, or while he was intent on grasping various objects. Three months after the accident, he had epileptic paroxysms, which did not respond to the bromide treatment. He was sent to an asylum, where on examination he was found to be somewhat stunted and a physical degenerate (high arched palate, slight asymmetry, face more developed than the skull, and scanty beard). The organs were in good condition. His mind was weak and he was highly irascible.

The myoclonus came on periodically but irregularly, and independent of surroundings. He might be free for a week, and then undergo a week of the movements. Examined in his calm periods, the nervous system, senses, reflexes, etc., seemed quite normal. The myoclonic attacks began suddenly, and as a rule in the second half of the night. The limbs were first involved, then the trunk and masseters, and occasionally the eyelids. The forearm was adducted and flexed; the arm was adducted and elevated; the shoulders were elevated; the thighs were adducted, and therefore when sitting the feet were raised from the floor; thighs were alternately flexed and extended on the pelvis; all segments of the lower extremity were extended, causing patient to fall forward or backward; finally the diaphragm was at times involved. All the muscles of the trunk and extremities were involved, only some of those in face escaping, as seen in Text-Plate C, Figure 18. These movements sometimes occurred isolated or in concert or predominated on one

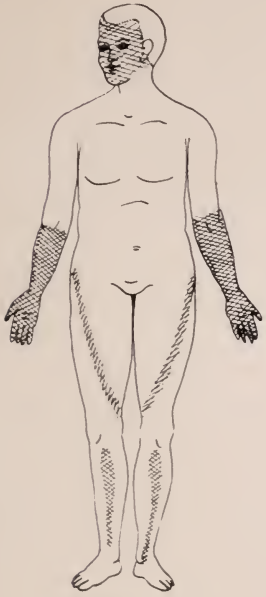


FIG. 13.
Seppilli's Cases.
Case I.

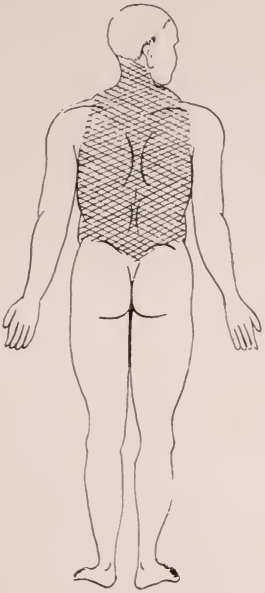


FIG. 14.
Seppilli's Cases
Case I.

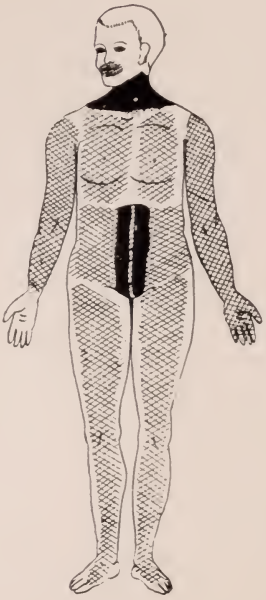


FIG. 15
Krewer's Cases.

FIG. 16.
Krewer's Cases.

FIG. 17.
Homen's Case.

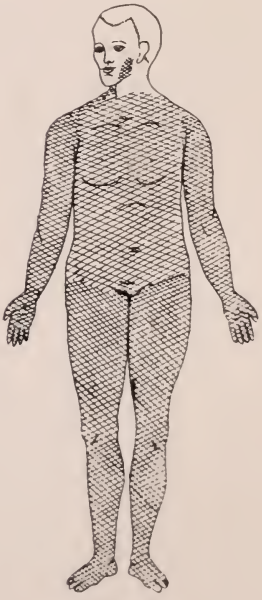


FIG. 18.
Garnier and Santenoise Case.

//=areas involved in epileptic fits.
::=areas most involved in epileptic fits.
#=areas involved in paramyoclonic spasm.
■=areas most involved in paramyoclonic spasm.

side. When recumbent, the extremities were involved, although in Professor Lemoine's cases* the contrary was observed. When the movements are at their maximum intensity, the intervals are shorter.

The will has a certain inhibitory power over the movements, so that the act of flexion might be largely arrested by the voluntary act of the extensor muscles corresponding. The movements are always absent during sleep. Very violent movements are affected by the will. The muscular spasms are painless, but induce fatigue. Percussion of the muscle, and sudden emotion, even if slight, aggravates the movements. Fibrillary tremor is absent.

The epileptic attacks occurred monthly. Great efforts were made to determine the effect of the epilepsy on the myoclonus. The latter was always worse before an epileptic attack, and there was no evidence of it for a day or two after.

One would have been inclined to doubt the diagnosis in Garnier and Santenoise's case, if the case had not been seen by skilled clinicians, and if the authors had not had the possible association of saltatoric spasms in epileptics well in mind.

Chapter VIII.

THE AUTHOR'S CASE.

Apparently this is the only case recorded by English or American observers. The patient developed epilepsy in early youth first in attacks of petit mal at the age of eight, changing gradually into grand mal. At this time after typhoid fever with a fall and severe fright the myoclonic spasms made their appearance. The epileptic

*I have not been able to find the cases of Professor Lemoine, who, on the authority of two French authors, is stated to have observed cases of association of paramyoclonus multiplex and epilepsy. The reference to Lemoine's observations are so indefinite that without consulting the original report it is impossible to state whether his cases should be considered in this paper.

attacks have become much better of recent years while the spasms have persisted. One epileptic fit seems closely associated with the myoclonic attack.

Case D. M.—male; age, 44; occupation, carpenter.

Family and personal history. A paternal uncle was insane, and had epilepsy produced by addiction to alcohol and morphine. Paternal grandfather died of abscess of the brain. A brother also died of "brain disease" at 38. Another brother is very neurotic but is able to do his work as postman. A maternal uncle died of tuberculosis. Father is very neurotic and intemperate and suffers from chronic rheumatism. Many paternal relatives were intemperate and neurotic without showing distinct diseases of the nervous system. Mother has always been troubled with sick headaches and two sisters of patient have had periodic attacks of facial neuralgia. A paternal niece had chorea.

Patient is the eldest living child. (An elder brother died soon after birth, of an unknown disease).

Patient had many attacks of cholera infantum in infancy and did not begin to walk until thirteen months of age. No illness of note until eight years of age, at which time he had a severe attack of scarlet fever.

A few months later, while fully convalescent, he began to have slight fainting spells (petit mal in character).

These increased in frequency and severity until in his thirteenth year he had typical grand mal seizures. At this time, following typhoid fever, he had a severe fright and a fall, in which he sustained a scalp wound. Immediately after this injury he was attacked with peculiar muscular spasms from which he still suffers. At periods of three or four years these spasmodic movements have almost entirely disappeared. His epilepsy has grown better of late years and he seems in a fair way to recover entirely from the epileptic attacks, not having had more than five or six seizures in two or three years.

One epileptic paroxysm which occurred while under my observation was as follows:

May 24, 1898.—The patient had a severe general convulsion of epilepsy. Previous to the attack he was more than usually involved in paramyoclonic spasm, especially of the muscles of the trunk and about the thighs. The seizure occurred while in the bath-tub. He knew of the threatened onset of the convulsion long enough before to cry out "I am going to have a fit." His voice was trembling, facial expression anxious and strained. Tonic convulsions came on immediately, extending over the entire body, and lasting about twenty-five seconds; this condition was finally followed by clonic spasms lasting about twenty seconds. Period of stertor lasted about three minutes. He was apparently unconscious for twelve or fifteen minutes after which he passed into a sound sleep for half an hour.

After recovery he was nearly free from contractures, able to walk about with ease without assistance, and to eat his meals without difficulty which he had not done before for three months. It was nearly one week before the muscles regained their former paroxysmal character.

Patient's periods of muscular tremor generally occur in the early morning hours, soon after rising. He has "good" and "bad" days, as observed in the other reported cases. The condition of the weather, excessive heat or cold, bright or rainy weather, influence the attacks for good or bad. There is no stammering nor difficulty of speech as a defect *per se*, but speech may induce spasms of the tongue or depressors of the lower jaw and is consequently short, jerky and broken. Mental state is very clear during the spasms, and notwithstanding the agitation and marked facial expression of anguish and apprehension he frequently laughs at his own misfortune and ridiculous appearance. Spasms are rarely attended by pain except in the extreme rotation of the head due to the twisting of the neck by the muscles of the left side. The muscular spasm is always bilateral and always of the nature of a shock, possibly a little more severe on the left side which at times appears to be, by the fraction of a second, the initiator in the spasm. He

often has difficulty in swallowing, food frequently being swallowed before he desires to do so. There is often a cramp-like feeling in the stomach after such acts. No muscles of either side escape the spasmodic movement, although those about the lower face, shoulders and thighs are most involved (seen in Figs. 1 and 2, Plate I). Occasionally patient's tongue thickens from side to side and many times it has rolled up like a ball in the back of his mouth. He has occasionally bitten his tongue, but never seriously. Generally the influence of attention and the exertion of will can stop or modify the spasm, but frequently excites it. Patient has received many severe injuries because of his spasms. He has fallen and broken his nose several times and received scalp wounds and contusions about shoulders and neck.

Spasms are most severe in the standing position, less in the sitting, and rarely present when lying down. Several times, while under observation during past few months, on attempting to rise from his chair, he has had a staggering and reeling period, finally ending in his falling heavily to the floor. These spasms begin by rapid protrusion and retraction of the abdomen until all the trunk muscles become involved. The spasms are absent during sleep and a severe seizure generally assures immunity for the next two or three days. He has no warning of the spasms. They generally occur in not more than fifteen or twenty distinct convulsive movements; then, by lying quiet he may be free for six or seven minutes, when they reappear, disappearing and returning in varying periods for several hours. Urine and fæces are never voided. Upon examination of urine no abnormalities were present and no indican was found.

Many self-inflicted wounds have occurred while using knives and chisels in his occupation as carpenter, his left hand being covered with scars. These wounds were inflicted through the spasmodic activity of the shoulder muscles and occasionally the biceps. He is not improving although for weeks his spasm may be confined to the deltoid and scapular region. He cannot rise; any

attempt to do so produces a general spasm and he falls heavily. For weeks at a time he has not walked without assistance. There are distinct spasms of diaphragm and pharyngeal muscles. He hiccoughs and produces sounds in the pharynx like the wood chopper or the workman lifting suddenly a heavy load. Superficial and deep reflexes are all exaggerated. Tapping for knee-jerks generally produce spasms. Co-ordinate movements lessen spasms.

Patient is of a religious disposition and obtains some freedom from spasms in the mental composure of prayer. The religious services of the Roman Catholic and Episcopal churches have a soothing and quieting effect on the spasm, while Methodist services are always disquieting. Bromides and chloral have some beneficial action for a time only. The hygienic and moral impress of colony life seems to have influenced his disease beneficially.

To summarize: This case shows bilateral paroxysmal spasms of trunk and shoulder muscles occasionally extending to the thighs and arms, but never in forearms, legs, hands or feet. They are always shock-like in character, unattended by mental disturbance. Attention is called to the experiments (Plate III) in obtaining ergograms of my case on "good" and "bad" days, and also the excellent showing of the voluntary inhibition in the patient's handwriting in Plate II which was taken on a "bad" day.

The ergographic experiments were undertaken to ascertain the modifying influence or effect of the administration of bromide and chloral upon the paramyoclonic movements. The results of the experiments, at three different stages of the observation, are given in the ergograms in Plate III.

The tracings were made by having the patient grasp a dynamometer first in a firm, steady grasp (tonic), thus keeping the muscles of the forearm and arm at their maximum volitional tension; and second, by having the

patient grasp the dynamometer repeatedly and as rapidly as possible (clonic movement) by his own volition. The patient was not otherwise instructed or coached while taking the tracings. The results are therefore thought to be fairly representative of the modifying influence of the two drugs upon the patient, and incidentally the tracings show the "good" and "bad" days of myoclonic spasm.

Chapter IX.

GENERAL REMARKS.

In conclusion, I would say that it is rather surprising, taking into account the character of myoclonus, that we do not see it more frequently associated with epilepsy. Its paroxysmal and spasmodic character shows its close relationship to this widespread affection of epilepsy. But from the mere fact that consciousness is so rarely at all disturbed proves that the difference in the underlying lesions shown in the play of the symptoms in the two affections is no narrow dividing line. Probably myoclonus is more closely related to the choreas pathologically, but of course this must be purely conjectural in our present knowledge of the functional nervous disorders.

While the diagnosis rests upon the triple complex of muscular symptoms,—paroxysmal, shock like and bilateral spasms of muscle; yet by looking over the different cases we find many variant cases reported under other designations which deserve to come under the head of myoclonus.

Although stigmata of hysteria are almost entirely absent in all cases, the majority of these cases are neurasthenic; my own was markedly so at different periods of his disease. I can also say that almost all the anatomical and physiological stigmata of epilepsy were absent in this case. As mentioned by Gowers, the use of alcohol gives immunity from paroxysms for a time in our case.

REFERENCES CITED.

UNVERRICHT, H.: Myoclonie. Lipzig und Wein, 1891, p. 128. Contains the original account of the first recorded cases. (Group of five).

UNVERRICHT, H.: Ueber familiare Myoclonie. Deutsch. Zeitschr. f. Nervenheilkunde, 1895, p. 32. Contains author's second series of (three) cases.

LUNDBORG: On Paramyoclonus multiplex och s. k. familiar Myocloni. Hygeia, Jan., 1899.

SEPPILLI, G.: Un caso di mioclonia famigliare associata all' epilessia Riv. sper. di freniatria, 1895, p. 373. Account of group of three cases studied at the insane asylum at Brescia.

KREWER: Paramyoclonus Multiplex Friedreich. Deutsch. Zeitschr. f. Nervenheilk., 1896, p. 62. Original account of author's case; also condensed account of Unverricht's first cases.

ZIEHEN: (Cited by Weiss—See Historical Sketch). Bare allusion to two cases seen by him.

HOMEN: Archives de Neurologie, 1887, p. 304.

GARNIER AND SANTENOISE: One Case of Paramyoclonus Multiplex Associated with Epilepsy. Annales Medico-psychologiques, 1898, VIII, 392.

DESCRIPTION OF PLATES.

Plate I.

Figure 1.—Shows anterior view of writer's case, D. M. Cross-hatched areas show parts usually and most involved; simple-hatched areas show parts least and infrequently involved. Epilepsy was classic, involving all the voluntary muscles of both sides equally.

Figure 2.—Shows posterior view of writer's case, D. M. Cross-hatched areas show parts generally and most involved; simple-hatched areas show parts least and infrequently involved.

Plate II.

The handwriting of the writer's case, D. M., written upon one of the patient's "bad" days. Patient is fond of United States history, and was asked to write an account of the battle of New Orleans, which is given here. The effect of volition is shown in the absence of myoclonic movement at the beginning of his description. Attention is called to the myoclonic movement in the words that are highly typical of action, or those that express energy, such as "strongly," "defended," "bloodless," "energetic," and the like. It will be noticed that the letters are all quite well formed and the muscles causing the myoclonus, as a rule, are those of the arm and shoulder, causing his handwriting to appear as though someone had struck his arm violently from behind while he was attempting to write the words. The wearing out, as it were, of his inhibitory power is well shown in the closing description of the battle.

Plate III.

Tracings of ergographic experiments on the case D. M. showing the effects of administration of bromides and chloral and the influence of "good" and "bad" days on the spasms. The dynamometer attached to an air tambour and connected with the kymograph was operated by the thumb and first finger. Some of the ergograms were traced by having the patient alternately grasp and relax the instrument uniformly and rapidly as possible by clonic movements. The remaining curves were recorded by a firm, steady and tonic grasp of the dynamometer.

I.—The first set of experiments, series 1 to 5 inclusive, was conducted before medication (bromide and chloral), and on one of patient's "bad" days, (May 28, 1899).

No. 1.—Shows the patient's volitional right hand grasp (tonic) at the maximum 24 k. The interruptions of the tracings mark the presence of more or less severe myoclonic movements over entire body, which find their expression in the patient's altered grasp. The interruptions always indicate spasm of the extensors, and are really negative manifestations of the spasm.

No. 2.—Shows the same test for the left hand. The myoclonic interruptions begin almost at once and continue to be much more frequent throughout the entire test than those shown in No. 1.

No. 3.—Shows the volitional right hand grasp (clonic), taken immediately after No. 2 (tonic), at 18 k. (Occasionally the little finger caught on the dynamometer in such a manner as to make a partial vacuum in the drum, which forced the tracer below the abscissa).

No. 4.—Shows the volitional left hand grasp (clonic), at maximum of 24 k. The myoclonic interruptions appeared later in the test than in No. 3; but the total length of time that the myoclonic movement existed during the tracing is about equal in 3 and 4.

No. 5.—Shows the volitional right hand grasp (tonic), at 26 k. Here we see the interruptions are greater and much more persistent than in No. 1, the first test of this period. As the patient had become too nervous and excitable to continue test, the effects of further experiments at one time were not practicable.

II.—(June 6, 1899). Patient had been placed upon bromide (grs. 180), and chloral (grs. 40), daily, since last ergograms were made, a period of nine days.

No. 6.—(A repetition of tracing No. 1). Shows the volitional right hand grasp (tonic), at 10 k., with but three or four slight myoclonic interruptions, in marked contrast to No. 1.

No. 7.—(A repetition of tracing No. 2). Shows volitional left hand grasp (tonic). The contrast existing between the two slight myoclonic interruptions in this case with that of the many in No. 2 is very striking.

No. 8.—(A repetition of tracing No. 3). Shows the volitional right hand grasp (clonic), at 20 k. But three or four slight interruptions occurred in this tracing.

No. 9.—(A repetition of tracing No. 4). Shows the volitional left hand grasp (clonic), at 20 k. The still slighter form of myoclonic interruption is shown in the slight indentations of the apex of the clonic curve. The tremor of the hands in the altered part of the tracings 8 and 9 show not only the muscular fatigue in these cases from exertion, but also the fine fibrillary (myoclonic) movement almost always present in true paramyoclonus.

III.—(June 11, 1899). This was taken after patient had been given bromide (grs. 180) and chloral (grs. 40) continuously from May 28, 1899, a period of fourteen days. Patient showed no myoclonic movements in performance of ordinary volitional acts, therefore this period typifies a "good" day of paramyoclonus; but even in "good" days the mental stress incident to unusual tests are shown in the recurrence of the myoclonic movement while the ergographic tracings were taken.

No. 10.—(A repetition of tracing No. 1). Shows volitional left hand grasp (tonic), at 17 k. One slight myoclonic interruption occurred during this tracing.

No. 11.—(A repetition of tracing No. 2). Shows volitional right hand grasp (tonic) at 17 k., and but one slight myoclonic interruption occurred at beginning of the tracing.

No. 12.—(A repetition of tracing No. 3). Shows volitional right hand grasp (clonic), and but one myoclonic interruption occurred throughout the test; otherwise the tracing appears normal, except slight tremor manifest near the end of the tracing.

No. 13.—(A repetition of tracing No. 4). Shows volitional left hand grasp (clonic), and but two very slight myoclonic interruptions occurred (marked X); otherwise tracing is normal, except slight (myoclonic) tremor, as seen in 4, 8, 9 and 12.

Fig. 1.

Fig. 2.

Gen. Jackson at New Orleans.

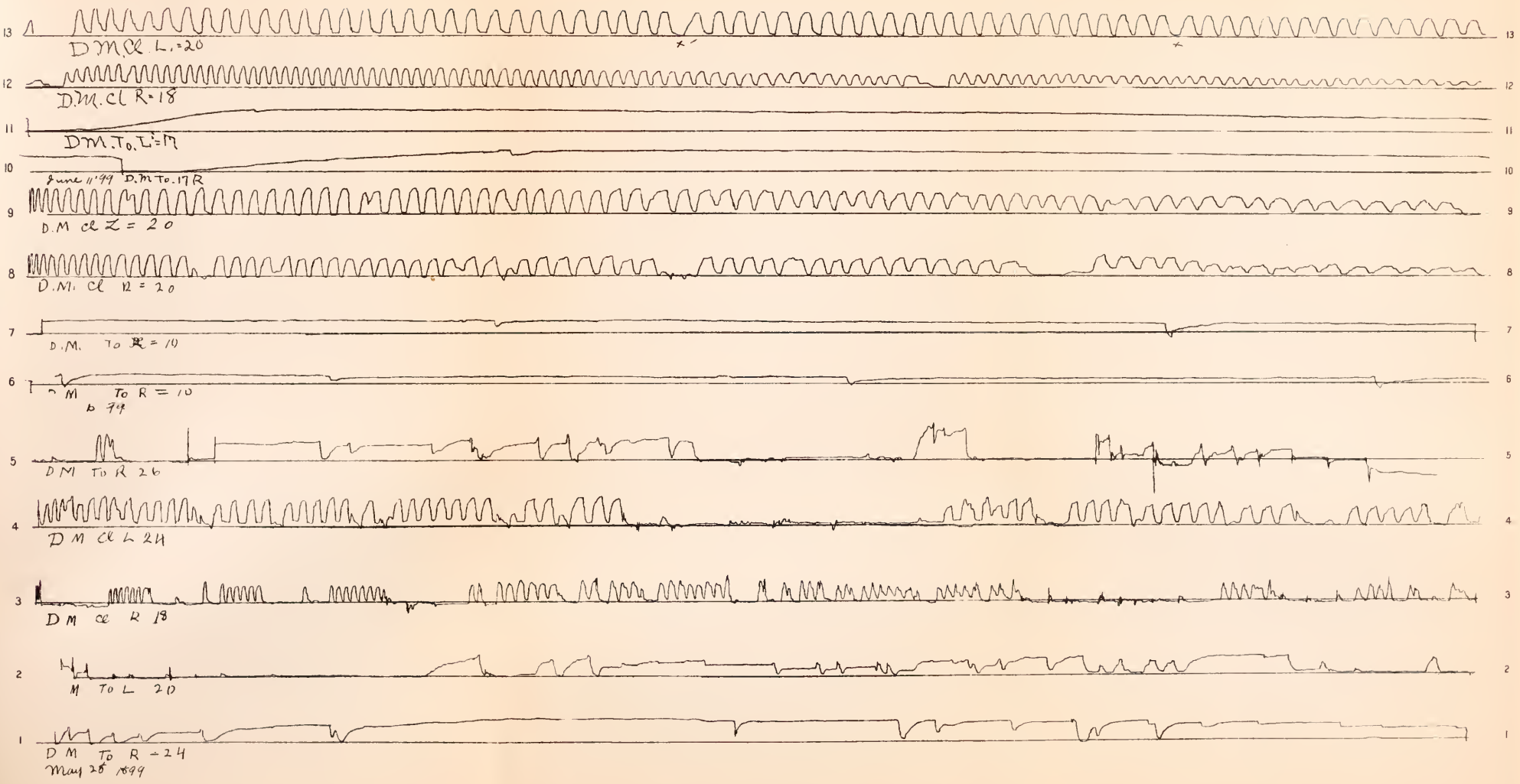
But what every schoolboy delights to read about and is one of the best known battles in our history was at New Orleans, when Jackson defeated the flower of the British army in the most sanguinary battle of modern times. Most of his troops were hastily gathered together as militiamen, who came with their rifles and fought on their own account behind breastworks. It may well be doubted whether Pakenham had any chance whatever of carrying the position held by Jackson, which was strongly defended; but even this does not account for the fact that the victory was almost as bloodless as that of Dewey's at Manilla or Schley's at Santiago. British troops have carried more difficult positions, but not against such troops. The American soldier is, first of all, a marksman. The American is the most energetic man in the world, but he hates to work for the mere sake of working. He must accomplish something. Whatever he does he does earnestly and intelligently. He goes to war not as a trade but to kill people.

(over)

The men from Tenesee and Kentucky with their long rifles were used to killing game for a living. Killing soldiers was natural to them. They shot to kill, whereas the British soldiers fired at the command of their officers. Nothing could exceed the bravery of the British men and officers at this battle. They stormed the American position repeatedly putting ladders up against the breastworks and doing all that men could do in the face of one of the fiercest storms of bullets that ever men confronted. But there is a limit to human endurance and the effort failed. It was much such a fight as took place recently at El Caney San Juan &c.

Now as Clarke human endurance is at an end with me also, so I stop

Munroe


13
DM Cl L. = 20
12
D.M. Cl R = 18
11
DM To. L = M
10
June 11'99 D.M To. 17 R
9
D.M Cl Z = 20
8
D.M. Cl R = 20
7
D.M. To R = 10
6
M To R = 10
b 7-9
5
DM To R 26
4
DM Cl L 24
3
DM Cl R 18
2
M To L 20
1
DM To R = 24
May 28 1899

III.

HYPERTROPHIC INFANTILE CEREBRAL PALSY AND PHOCOMELUS ASSOCIATED WITH EPILEPSY.*

INTRODUCTION.

In the study of epilepsy it is desirable to approach the subject from as many points of view as possible in order to comprehend that class of cases known as idiopathic epilepsies. In studying the disorders of motility in epilepsy, with their possible relationship to subsequent paralytic states from exhaustion, and in tracing the association of paramyoclonus with epilepsy, it is very interesting to note the effects of chronic convulsive disorders (such as epilepsy, athetosis and post-hemiplegic mobile spasm) on the growth and development of the epileptic physique. It is also highly important for us to know all the successive links existing between focal (Jacksonian) and classic epilepsy. In order to do this, it is essential that we should study many cases of spasmodic infantile hemiplegia, where epilepsy exists as one of the many symptoms of the gross cerebral disease. Largely with these ends in view, this paper is presented in these clinical studies in epilepsy.

The subject of the growth and development of the human body still remains in the domain of mooted questions, but laws governing the increase in size and weight of man in different races and in various environments have been more or less carefully established by anthropologists and physiologists, and are fairly well known.

* Read in abstract before Section on Medicine, Buffalo Academy of Medicine, November 8, 1898.

The different degrees of atrophy, from its marked presence to the point where it is but little manifest at all, in the so-called essential palsy of children or infantile spinal paralysis, have been well studied. Notwithstanding marked atrophy is the rule, Seeligmüller has described some extremely rare cases of actual elongation of the bones in infantile spinal palsies, which is attributed to the growing epiphysis suffering traction instead of the usual compression. Dejerine claimed to have demonstrated a true muscular fibre hypertrophy in muscles totally paralyzed, although it is extremely doubtful that any muscle can be so paralyzed that some individual fibres may not undergo voluntary contraction (Gowers). However this may be, muscular fibres in infantile spinal palsies have been proven larger than normal in a few selected cases, even forty or fifty years after the acute onset of the spinal palsy. This was especially shown by the researches of Joffroy and Achard in 1889.

Although the positive exclusion of some nerve cells from the initial damage in the anterior horns of the cord might possibly explain the isolated phenomenon of muscular fibre hypertrophy, yet such theories can hardly hold a place in the exceptional instances of hypertrophy in the infantile *cerebral* palsies. The complete annihilation of the theory that there are special trophic nerves and centres renders the attempt at explanation of hypertrophy much more difficult.

The presence of normal muscles and bones of the paralyzed parts of an individual suffering from cerebral palsy might be understood in part; but why the once paralyzed parts should actually become hypertrophic is mysterious in the extreme. The mobile spasm or athetotic theory has not a sound basis of fact, since cases hereinafter cited

from literature, as well as one case reported by myself, show that the theory must be discarded.

The athetotic theory which, in short, accounts for the hypertrophy of paralyzed parts by continued overaction of these parts in mobile spasms, should, if true, hold in other disorders of non-volitional motility—such as chorea, *tic convulsive*, and even epilepsy. As has been mentioned in another part of these clinical studies, one must carefully avoid the erroneous conclusion that the results of physiological and pathological expenditures of energy are the same. The absurdity of such conclusions is apparent to all on close examination. Even supposing that autopsies upon cases illustrating the hypertrophic forms of infantile cerebral palsy were to be had, the colossal molecular changes which are necessary to make the physiological pathology of the ordinary functional neuroses perceptible to our present advanced methods of microscopical study, are so very great that there would be but little hope of our discerning the real cause of the hypertrophy in these infantile palsy cases. Possibly when we shall have attained to some definite knowledge of the etiological pathology of athetosis, we may find some cause for the rare instances of hypertrophy of paralyzed parts in infantile cerebral palsy cases, as athetosis is present in the majority of them. Such remarks cannot be held to militate against the writer's position in this paper, as the positive proof of the absence of *one link* (a case without athetosis) in the chain of the athetotic theory renders it void.

I can find no special article in medical literature upon the subject of hypertrophy in infantile cerebral palsy. No such case has been reported in America, and I doubt whether one has been seen. Sachs and Peterson, in their extensive acquaintance with cerebral palsy cases have

apparently not observed such a case. The great amount of literature upon infantile cerebral palsy is devoted almost entirely to patients while still in infancy, when we cannot expect to find this hypertrophic condition, as some years are probably required for its development.

Most of the cases of hypertrophy are described under the head of athetosis. In going over the literature of the subject of athetosis and infantile palsies, we find seven cases described in detail. Two of these cases are inaccessible. I shall give abstracts from five of the cases and then append in full two cases of my own, which are the only ones on record associated with epilepsy, although we find epilepsy in the *family history* of other reported cases.

Chapter I.

HISTORICAL REVIEW OF THE LITERATURE.

Hammond, who first discovered and described hypertrophy in connection with athetosis, makes no mention of any case dating from infantile palsy or indeed from childhood at all. The same may be said of the next half a dozen of writers (1871–1873).

Clay Shaw, in 1873, first described athetosis which dated from childhood, and gives a number of histories. He was on the lookout for hypertrophy and says expressly that it was not present; he says of one or two of his cases that the "muscles of the affected side were firm."

Bernhardt is said to have observed the first case of hypertrophy in connection with infantile hemiplegia in 1874, although I find it reported in 1876. During this year he reported a second case. Gowers also in the same year reports one and perhaps two cases, although I think that the second of his cases is merely due to a clerical error.

In 1878, Dreschfeld of Manchester published a series of cases similar to Shaw's, and says that atrophy was invariably present. In the same year Oulmont published his thesis on athetosis and gives many histories, in one of which hypertrophy is described in connection with infantile hemiplegia. In 1881, Ross in his work on nervous diseases, speaks of Shaw, Dreschfeld and Oulmont as the leading authorities upon athetosis dating from infantile cerebral disease.

Since 1881 abundant material has been presented upon cerebral palsies from many writers, but the question of hypertrophy is either ignored or it is expressly stated that atrophy was present. Only at long intervals do we find any mention of cases, or indeed of hypertrophy as a fact possible. Osler's extensive study, for example, is silent upon the subject (1882). Gaudard, writing in 1884 on infantile cerebral hemiplegia, gives many histories including one with hypertrophy.

Berger, in his article on athetosis in Eulenberg's Encyclopædia (1885), speaks of hypertrophy as present in a small per cent of cases of athetosis of adult life, while in cases which date from childhood we find a large contingent of atrophy. He then seems to imply that hypertrophy dating from athetosis of childhood is an inexplicable freak of pathology.

The next case in literature is by Scheiber, published in 1890. Since this case was reported there has been a period devoid of new material. Freud and Rie who published an extensive study of infantile hemiplegia in 1891 add nothing new. Brissaud, in 1894, writing on infantile encephalopathies for Charcot's Traité de Médecine, ignores the subject, while Sachs, in this country, in his work on Nervous Diseases of Childhood (1895) says expressly that

while he has been on the lookout he has never seen a case of hypertrophy.

This brings us to 1897, the date of Freud's voluminous monograph on infantile cerebral paralysis, which constitutes a separate volume of Nothnagel's Specielle Pathologie und Therapie. In this book is found the first separate treatment of the subject of hypertrophy, occupying less than a page. As it gives a fair summary of the subject, we reproduce it entire (p. 95).

"As for the interesting symptom of hypertrophy, it cannot be denied that it is intimately related to the violence of the spontaneous movements (athetosis). Moreover, it is found in cerebral palsy of childhood only when the athetosis is very well marked, as in Gaudard's case, in both of Bernhardt's cases, in Oulmont's and in several of Gowers'.

"In Bernhardt's first case the hypertrophy accompanied a primary athetosis (choreatic paresis); in the second it accompanied a late chorea (post-hemiplegic athetosis). In Bernhardt's second case, hypertrophy was present only in the lower extremity where the spontaneous movements were more violent than those of the arm. In Oulmont's case the arm was hypertrophied, the leg atrophied, corresponding to the fact that the arm alone was affected."

"Scheiber's case gives an interesting combination: In a case of hemiplegia there was an arrest of growth in the entire left half of the body, with hypertrophy of muscles of left arm."

Oulmont states the case for hypertrophy and atrophy as follows: "If paralysis is slight or transient, nutrition remains intact. If athetosis sets in, the muscles must necessarily become hypertrophic. If paralysis was severe or prolonged, atrophy alone can result."

"This, however, can hardly be the case, for if it were so, hypertrophy would always occur in every severe case of choretic paresis; whereas it is very infrequent in this condition. On the other hand atrophy can occur in a pure

choretic paresis. It is therefore justifiable to assume that the alteration of nutrition is a symptom which is independent of the cerebral lesion."

Since the publication of Freud's monograph, there has been a case reported by Kaiser which is in some respects more interesting than any of the preceding, the degree of hypertrophy being greater, while, according to the author, myotonia was for the first time present as a complication. This case is also unique in showing that hypertrophy of the bones may occur in these conditions as shown in the skiagraphs of our own cases. We will now collect these scattered cases, give them in abstract, and then detail our own cases.

Chapter II.

BERNHARDT'S CASES.

Bernhardt reported two cases at about the same time, but in separate articles. All authorities give him credit for reporting the first case in 1874, although it will be seen that both his cases are reported in 1876, the same year in which Gowers wrote.

Case 1.—*Family History.*—Male, age 12. Possible family taint. One brother acquired after extraction of a tooth a condition of unrest in all the muscles. Improved under constant current, but became much worse after the root of the tooth was extracted, and death soon followed (age 16 years). A sister had well marked chorea at age of 10 years, but after six months recovered completely. Rest of family of parents and twelve children have good or negative history.

History of Stroke, etc.—Was well until fourth year when was confined to bed for several weeks by an acute affection which was probably one of the exanthemata. After this affection a false position of the right foot was observed.

History of Athetosis.—Abnormal movements of the right hand were present from the first, that is as soon as there was any evidence of hemiplegia.

Measurements.—The right forearm, immediately above the wrist, measures 14.2 cm.; the left, 13.3 cm. The right forearm at a point 11 cm. below the internal condyle measures 16.3 cm.; the left, 15.8 cm. The right upper arm at the middle is 17.5; the left 17.2. (For comparison of athetosis and hypertrophy, see Text-Figure 1). The whole right forearm is thus more voluminous than the left, and this *despite the fact that the patient learned to use the left arm for all purposes*, since the involuntary movements made it quite impossible to depend on the right. Involuntary action in this case would seem to be a more potent cause of hypertrophy than voluntary.

Posture, Gait, etc.—Nothing is said as to posture of the upper extremity, probably because the shoulder and elbow joints are normal, and the peculiar carriage of the arm and elbow noted in other cases is wanting here.

Posture of Foot at Rest.—As regards gait, patient walks good distances although at times the right foot takes an equinovarus position with toes turned toward the ground. The right leg is fully quiet at hip and knee, but there is a slight persistent varo-equinus position of foot, tension of tendo Achillis and distinct plantar flexion of toes.

Movements.—Hand.—If the patient sits quietly with attention diverted, the right hand is fully quiet. When attention is directed to the patient there is a remarkable change in the movements of the fingers. In quite rapid succession they are ad- and ab-ducted, flexed and extended, especially the thumb, index and little fingers. Although the hand is quiet there is a distinct play of the flexor and extensor muscles under the skin.

At times, but to a much less degree than the hands, the toes move upward and laterally. It is said (contrary to the usual experience) that the movements of the toes are worse at night.

Condition at Large.—Intelligence good, frequent headaches, special senses normal. Inequality of two sides of

face, at rest and in action, but equal in sleep. Muscles on right side act more slowly and are weaker. At times pain in whole right lower extremity. Lowered sensibility on right side.

Disease has persisted unchanged seven years. All treatment unavailing. By a strong effort of will patient can, sometimes, arrest movements.

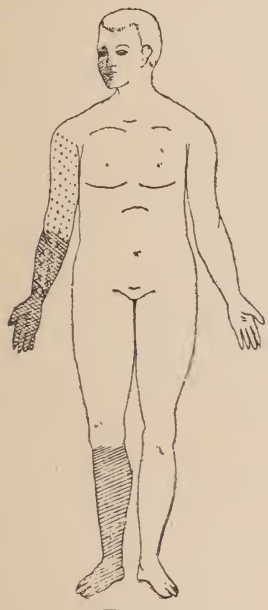


FIG. 1.
Bernhardt's 1st Case.

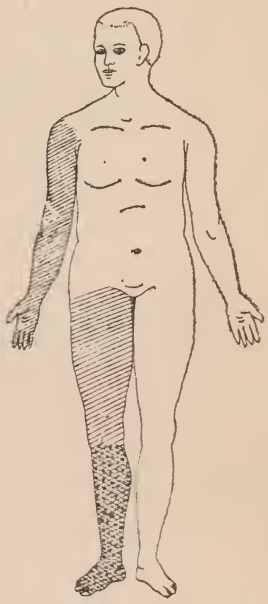


FIG. 2.
Bernhardt's 2d Case.

//=parts showing athetosis.
##=parts showing most athetosis.
⠿⠿=parts showing hypertrophy.
××=parts showing most hypertrophy.

CASE 2.—Male, age 14 years. Family history good. Patient healthy until fifteen months of age.

History of Stroke.—It occurred at night at age of fifteen months. No further details.

History of Athetosis.—This is not given.

Present State.—Measurements.—No figures given.

Simply states that the right (or paralyzed) leg is larger than the left, while the left arm is larger than the right.

General Character of Movements.—Active movements of all sorts are possible with the right arm but they are slower, weaker and more uncertain than those of the well side; this is especially true of the fingers. When the right hand and fingers are supported they remain quiet for a long time, longer than do the foot and toes. Occasionally a jerking of the entire right upper extremity is observed, but as a rule shoulder and elbow do not move.

Posture, Gait, etc.—When the right arm is held free, the forearm at once becomes prone, hand flexed towards volar and ulnar surfaces, fingers moving in all directions (especially thumb and little finger). Patient can walk alone, but drags his right foot a little, foot being flat and in valgus position. If the legs are close together and supported beneath, the right foot takes a varo-equinus position which persists. Toes move incessantly much more than the fingers, both toward the dorsal and plantar surfaces of the foot. (See Text-Figure 2 for athetotic and hypertrophic conditions).

General Condition.—Robust physically, weak mentally, cannot read or do simplest sums.

GOWERS' CASES.

Freud says that Gowers reports several cases of hypertrophy dating from infantile cerebral disease. After a careful review of the eighteen cases described by Gowers, I find that seven began in childhood, the oldest being nine years old. In the first of these seven it is expressly stated that hypertrophy was present; in the second, "the paralyzed arm was of the same size and as well nourished as the other;" in the third, atrophy was present; in the fourth, the affected arm was a little smaller and there is no mention as to the leg; in the fifth, there is no mention of size; in the sixth, "arms of same girth;" in the seventh

and last, the right (affected) arm is much smaller than the left, and it is then stated that the "left leg is *also* much smaller than the right." This is so obviously a clerical error that it could not be mistaken for a case of hypertrophy, so that unless Freud regards the second and sixth cases, where the arms were of the same girth, as examples of relative hypertrophy, I cannot see how Gowers can be said to have reported more than one case, which is here appended.

GOWERS' CASE III (OF WHOLE SERIES).

Mobile and fixed spasm of left arm with musular hypertrophy. Overaction of facial muscles. Sequel of infantile hemiplegia.

Female, age 29.

History of Stroke.—Came on at age of six years while suffering from scarlatinal dropsy. Left hemiplegia with convulsions. Probably complete paralysis with slow recovery of power. Few details obtainable. (Text-Figure 3).

History of Athetosis.—Spasm of arm a year or so after stroke. Arm bent at elbow, forearm flexed so strongly it could not be straightened, fingers and wrist flexed. This posture maintained for years, then suddenly changed, the forearm became extended, while the fingers remained flexed, the whole arm carried backward behind the trunk. This posture persisted until age of 25 or 26, when she became able to flex and extend the forearm at will, although the spasm persisted in the meantime.

Present Condition (June, 1875).—*Measurements.*—Left arm shorter than right, left ulna measuring nine inches, right ulna ten inches. Nevertheless left arm thicker. Maximum circumference of left forearm nine and a half inches, to nine and a fourth on the right side. Increase in size due to muscular hypertrophy.

Posture, etc.—Muscles in condition of continuous but varying spasm. The common position of the arm is by

the side with elbow extended, wrist and fingers flexed, but in a varying manner, the ring and middle fingers being flexed at the metacarpo-phalangeal and also at the middle joints; while the index and little fingers are flexed only at the former, being extended at the two phalangeal joints (interosseous position). The thumb is overextended at all joints.

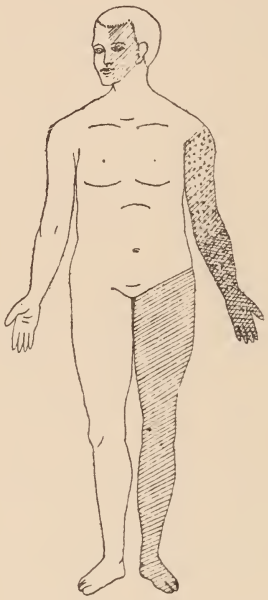


FIG. 3.
Gowers' Case.

//=parts showing athetosis.
##=parts showing most athetosis.
⠿=parts showing hypertrophy.
⠿=parts showing most hypertrophy.

Movements.—Upper Extremity, Fingers.—The position of the fingers changes, the thumb is often pressed so as to press back the last phalanx. At other times the first finger is extended at all joints, the others flexed at the metacarpo-phalangeal joints. Fingers occasionally overextended at middle joints. It is with difficulty that the fingers can be

held still enough for photography. Patient has little power of arresting the movements which go on slowly but continually. When the hand is at rest for a few moments, the spasm becomes much slighter; patient can then with the other hand extend the phalanges completely and almost extend the wrist. A voluntary effort brings back the spasm in all its uncontrollable intensity. When patient is asleep the hand is always still and probably limp. During sleep the arm is at times at her side, at times across her chest. Wrist flexed at times in sleep.

Lower Extremity.—The leg is much stronger than the arm. It was originally turned as she walked on it, but improved as she gained strength. She can now walk a fair distance but the foot is always a little inverted and the toes turned up, and this condition is increased by any attempt to move the foot. Text-Figure 3 shows parts most in athetosis and their corresponding hypertrophy.

Condition of other parts.—Gowers describes in full the marked inequality of the two sides of the face at rest and in action. The general condition appears to have been good. This case is illustrated by a number of small cuts.

OULMONT'S AND GAUDARD'S CASES.

As the authorities wherein the two above cases are to be found are not accessible in the original, we have only the very brief citations of Freud, who says:

"In *Oulmont's* case the arm was hypertrophic while the leg was atrophic; corresponding to the fact that the arm alone was affected (by athetosis)".

Of *Gaudard's* case Freud says merely that "the athetosis was of great intensity."*

* It is to be regretted that the references to the two cases just cited are so incomplete, in not stating parts most athetotic and hypertrophic, that text-figures can not be made to help us in forming a complete picture of the relationship existing between athetosis and hypertrophy in the whole series of cases cited in this paper.

SCHEIBER'S CASE.

CASE I.—*Family History.*—Male, aged 16, (schoolboy); possible family taint—father had headaches and was irritable; an infant brother died of eclampsia; patient also suffered with infantile eclampsia. Well up to seventh year excepting a right purulent otitis media with mastoid abscess and subsequent deafness at five years.

History of Stroke.—In seventh year a seizure at school; fell from seat in a sort of swoon, but did not lose consciousness although sight and hearing were obscured. Complete left hemiplegia followed with aphasia. Intelligence preserved; could not write and make wants known. Was confined to bed for five weeks and vomited at times with dribbling of saliva.

History of Athetosis.—At the expiration of the five weeks' sickness, spasms appeared in the left arm and leg which have since persisted as tonic spasms.

Present Condition.—Measurements.—The muscles of the left upper extremity are hypertrophied and especially the muscles about the shoulder joint, (trapezius, supra-, infra- and subscapularis, and deltoid, and the other arm muscles). Circumference of upper arm at middle, and of forearm at thickest portion measures 1 cm. more than on the well side. In spite of this hypertrophy the left side is no stronger than the right. In the lower extremity the conditions are reversed. The left muscles are somewhat weaker and the left calf at its thickest portion is 1 cm. less in girth than the right. This is probably due to the fact that the tonic spasms in the leg are far less intense and bear a direct relation to the hemiatrophy, while in the arm the much more intense and almost ceaseless tonic spasms cause a hypertrophy despite the tendency to hemiatrophy.

Movements in General.—Spasms are very well marked in the wrists and fingers, and in a much slighter degree in the elbow and shoulder. In the lower extremity, excepting the hip, the same spasms occur more marked in the toe and ankle than at the knee. Spasms are purely

tonic and persist in individual joints from ½ to 3 minutes. Great force does not suffice to overcome the spasm which subsides spontaneously.

Posture, etc.—Fingers sometimes in hyperextension, at others clenched with the thumb either in the palm or pressed against the side of the index finger. When the thumb is in the palm the spasm ends sooner than otherwise, for when it is pressed against the side of the index

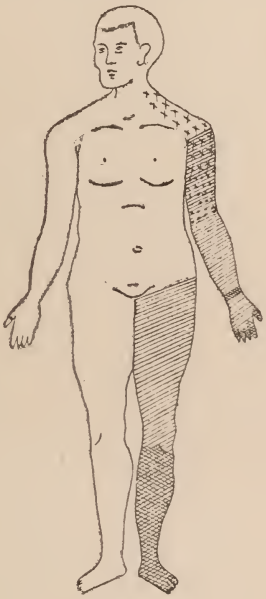


FIG. 4.
Scheiber's Case.

//=parts showing athetosis.
##=parts showing most athetosis.
⁘=parts showing hypertrophy.
××=parts showing most hypertrophy.

finger it retards opening. In extension spasm, all fingers participate as a rule, but at times only one or two are extended, the rest being semi-flexed or wholly flexed. It often happens that one or all of the fingers are in adduction or the reverse.

The *wrist* is flexed either in dorsal or volar posture, but seldom in ad- or abduction.

The *hand* is usually in pronation, often in hyperpronation, rarely in supination.

The *elbow*-joint is chiefly in extension, rarely in flexion.

Combination Posture.—When the patient is on the street or excited, the whole upper arm is extended backward. Originally the arm used to make an angle of 90 degrees with the trunk, but this has improved so much that at present the angle is but 30 degrees. With the arm in this posture, the hand would be in hyperpronation, wrist in dorsal flexion, fingers clenched. This combination occurs much less frequently than at first.

Lower Extremity.—In the lower extremity there is more monotony. At the knee there is only extension. At the ankle, pes-calcaneus or varo-calcaneus. Toes held both in dorsal and plantar flexion; the most frequent combination is great toe in the dorsal and the others in the plantar position.

Special Movements.—As the tendency is toward rest when the patient is quiet, whatever disturbs or excites develops the movements. Among these perturbations are attempts to use the well hand, such as going out in the street, and in general whatever produces self-consciousness. The finger spasms last as long as the patient is watched or thinks he is being watched, or as long as he is occupied with his other hand, or is being spoken to, or until his temper changes (as from grave to gay, etc.)

Lower Extremity.—Spasm of foot and toe impedes but does not prevent locomotion. With knee spasm patient has spastic gait. Spasm in lower extremity does not last as long as in the upper; spasm of shoulder and elbow not as long as hand and fingers.

Patient unable to act upon the movements by will power, in fact this will aggravate them by voluntary motion of the well hand. There is some slight effort possible in overcoming extension and causing the fingers to return to flexion. (See Text-Figure 4 for parts athetotic and hypertrophic).

Condition at large.—Scheiber describes very fully the state of hemiatrophy and the degree of motor and sensory hemiplegia which co-exists.

General health and intelligence good.

KAISER'S CASE.

Male, 19 years. Family history negative excepting that one brother is an epileptic. When seven years old developed left-sided hip disease. In hospital two and one-half years, chiefly abed, lying on right side. No active use of right arm, but much used for supporting the body.

History of Stroke.—Was not conscious of any stroke at any time. Only when he left the hospital he found his right arm permanently affected.

History of Athetosis.—Began as soon as he left the hospital and went about; involuntary movements of right arm and fingers and right toes but not enough to prevent walking.

Present Condition.—Measurements.—Left arm and leg normal, except that left thigh is somewhat atrophic from hip disease. On the right or affected side all the muscles are in athetotic condition, including the pectoralis major, latissimus dorsi and trapezius (Text-Figure 5). Muscles firm, and when contracted, hard. Even the bones are hypertrophied, the right hand being better developed than the left. The right hand is cyanotic.

The right upper arm measures 24 cm. to 22 of the left; the right forearm is 23½ cm. to 22½ in the left. The right hand measures 22 cm. around, the left 21. Length of right middle finger 11½ cm., of left 11 cm.

The right calf measures 31½ cm., the left 30. The right foot measures 25½ inches around, the left 24. The results of hip disease prevent comparison of thighs.

General Character of Movements.—Tonic spasms are present when patient's attention is directed to himself or to some voluntary motion. When attention is diverted, the arm is again relaxed. Voluntary relaxation is impossible.

With this patient the athetotic movements are combined with myotonia (so Kaiser thinks). In myotonia if the patient presses a pencil *voluntarily* against his right palm with his finger, he is unable to relax his finger (extend it) for at least thirty seconds.

The athetosis in this case consists of a very slow inward rotation of the right arm, and an extension backwards, upwards and outwards, shoulder being slightly raised.

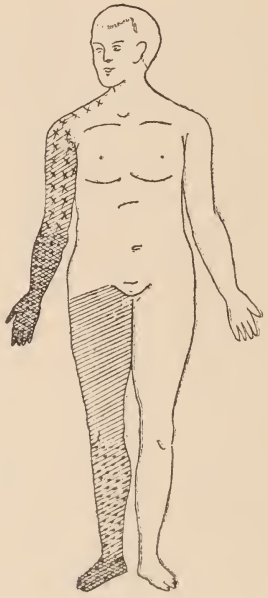


FIG. 5.
Kaiser's Case.

//=parts showing athetosis.
##=parts showing most athetosis.
⠿=parts showing hypertrophy.
⠿=parts showing most hypertrophy.

Sometimes the forearm is flexed at an acute angle, hand extended, fingers slightly flexed. In the right leg athetosis is best seen while walking. As soon as a step is

taken the motions begin. Toes are so strongly flexed that the nails touch the ground, inner edge of foot raised and a varus position results. After this position is maintained for a time, toe movements begin, mostly in extension. While at rest athetosis movements in the perineal and tibial region are seen, which does not occur while walking. (See Text-Figure 5).

Author has discussed at considerable length the phenomenon of myotonia in this case. It is so extremely difficult to differentiate the very slow athetotic spasm in some cases of athetosis from pure myotonia that it is doubtful if Kaiser's case really represents the previously unknown association of athetosis and myotonia. Probably the lack of movements in this case after position of parts are fixed at any given point are due to the so-called slow rhythmic athetosis.

Chapter III.

THE WRITER'S CASES OF PHOCOMELUS WITH EPILEPSY.

In connection with hypertrophic states of infantile palsies discussed in this paper, it is of considerable interest to refer to diminutive or dwarfed humeri as illustrated in two cases of phocomelus of the humerus, which have been classed by the author as degeneration-stigma.* Phocomelus of the humerus is a very rare condition, there being but possibly one other case reported (Raymond and Janet †).

The variation of the length of the humerus is considerable, even in what is considered within normal limits—one-half or three-quarters of an inch. Hence such normal

*Clark: Phocomelus of the Humerus in Epilepsy as a Stigma of Degeneration. New York Medical Journal, May 13, 1899.

†Raymond and Janet: Malformations des mains en pinces "de homard" et asymmetrie du corps chez une epileptique. Nouvelle iconographic de la Salpêtrière, No. 6, 1897.

variation would not be a degeneration-stigma. Raymond's and Janet's case was but one centimeter.

In regard to phocomelus in general, it can be said that it rarely occurs except in monsters, and monsters rarely live any length of time. Phocomelus of the humerus is rarer than the same affection of other long bones. In fact the humerus seems to suffer abnormalities much less frequently than other long bones. Tonnino* and Zuccarelli have done the most work upon monstrosities in connection with epilepsy of any investigators of recent years. Zuccarelli's work was done in 1885, and is not of so much importance as Tonnino's from the standpoint of epilepsy. Many recent writers upon neurology seem to have a prejudice against considering abnormalities as stigmata (notably Sachs and Oppenheim) and consequently the subject is still frequently imperfectly understood because of improper presentation.

The cases of phocomelus, including measurements, photographs and skiagraphs, are as follows:

Case 1 (Plate I, Fig. 1).—L. S., a girl, aged nineteen years; of Irish parentage, single, and of no occupation. Has had epilepsy since three or four years of age (exact year unobtainable). Father intemperate and mother had rheumatism. Patient is seventh child in family of three girls and four boys. Mother died of phthisis at forty-one years; father died of unknown disease at fifty-three.

Patient was a full-term child; delivery was instrumental, and labor was prolonged. Patient was injured on left side (probably birth palsy) and had several convulsions after birth. She was subject to "fits of crying spells" from infancy, and when dentition began at three months of age she had great difficulty in digestion with malassimilation. Several convulsions occurred at this time also.

* La epilessia in rapporta alla degenerazoni, Torino, 1891.

She began to walk at fifteen months, but suffered for two or three years of early life from suppurating scrofulous glands in the neck.

When about three or four years or age she had several severe convulsions which ended in left-sided hemiplegia; she was afflicted also with mobile spasm of an athetotic character. The second attack occurred about two months after the first. Her attacks for many years were confined to the left side, and always began there. She occasionally has partial epilepsy, which occurs as follows:

Four attacks of the same general character occurred April 6th, 1898, while at the Colony. Attacks began with convulsions in left side of face, unattended by the epileptic cry. Her head was drawn to the left, then the tonic spasm was changed to clonic spasm; eyes were wide open and staring, while pupils slowly and widely dilated. The hands were not clenched or even closed during the attack. Convulsive movement was confined to the left side of the face. Following the convulsion copious frothing at the mouth took place. Unconsciousness followed the convulsion and lasted but a few seconds. Her seizures are generally of the grand mal type, mostly confined to the left side, although usually general after the invasion is complete. Her attacks are almost always preceded by epigastric aura, a fact fully recorded by Gowers, viz., that hemiplegic epileptics may, after a time, develop auræ similar in all respects to those occurring in idiopathics. She has had as many as seventeen fits in twenty-four hours.

Physical examination showed a general poor physique; body poorly nourished; left internal strabismus; pupils dilated; left pupil larger. Breath was foul; she was constipated and showed marked symptoms of bromic poisoning. All deep reflexes were exaggerated on left side. Right arm was in all respects normal except as to length. Measurements are given at end of abstracts.

We must remember that we are not comparing the shortening of this humerus with a normal one, but with a defective development of a left hemiplegic of three or

four years of age. The comparison would be much more striking if it were as in Case II, to follow. Both patients are ambidextrous and equally strong right or left. The physiological compensation of throwing the right shoulder forward to make up the shortening of the right humerus is present in both cases.

The mobile spasm in this case at times closely resembles true athetosis, as is so well described by Hammond and illustrated by Strümpell. Intentional movements of the hemiplegic extremities are always overacted; this is also apparent even in the face in emotional and volitional acts. Athetoid movements are most typical in the thumb and big toe of the left side. Notwithstanding athetoid movements are extreme in this cerebral palsy case marked atrophy is present. The patient presents the customary mental weakness of a hemiplegic and epileptic. It was impossible to obtain a skiagraph of this interesting case as patient invariably broke the plate because of mental and physical agitation.*

Case II.—A. A., (Plate I, Fig. 2. Plate IV, Fig. 2) a woman, aged 20 years; nativity, United States; single; no occupation. October 6, 1898, she was admitted to Craig Colony suffering from epilepsy dating from eighteen years and a half.

Family History.—Aside from deaf-mutism in a paternal uncle, and consumption and rheumatism in mother, the family history was good or negative. The patient is the sixth child in a family of seven, five girls and two boys. The two boys died; one of diphtheria at two, the other of cholera infantum. No history of trauma or complication during her mother's pregnancy. Patient was born at full term, and was a healthy child. No instruments were used, and labor was not prolonged. Nothing unusual of

* Case L. S. had an attack while under the X-ray. The convulsion began in the left arm which was raised and thrown across the chest toward the right shoulder. There was no movement in the left leg or foot; slight clonic spasm was noticed in the whole right side some few seconds after the attack began in the left arm. There was no stertor and patient remained automatic for a few seconds after the convulsion. Patient complained that the X-ray gave her "pins and needles sensation" on the right side of the chest. It is probable that this latter sensation was from the electrical machine.

right side was noticed until two years of age, when the knee became very painful, swelled, and she was unable to use it. After a period of several months to two years contracture and rigidity necessitated a tenotomy of the Achilles tendon; a year after this operation another was necessary and an amputation just above the knee was done. This condition was in all probability tuberculosis, from the relatives' description. The right arm became an object of attention at this time, and the surgeon said probably the arm was paralyzed once. This explanation proved convenient until the family physician said rightly that such an explanation was impossible in view of the fact that the patient still maintained such perfect use of the limb. No further explanation or comment was made until she was brought to my attention at Craig Colony, when, from the examination of the case, I judged her worthy of a somewhat lengthy report. Patient has one sister who is of short stature; aside from this, no such phenomenon was ever present in the family, even if this fact has any bearing on her abnormity.

History of Epilepsy.—Her epileptic seizures at first were grand mal type, but generally convulsive, occurring every two or three months; now they are lighter, of petit mal type, but generally convulsive, occurring every five or six days. She has never had any warning before fits, which generally occur in the early morning hours. She is bright, and capable physically, notwithstanding she was not able to learn at school, and uses crutches to get about.

Physical Examination.—Physical examination mostly negative. Of good physique; nutrition well maintained. Left pupil was found much larger than right; vision was poorer in left than in right. Superficial and deep reflexes apparently uniformly normal. On examination of the shortened humerus it was found that the condyles were much enlarged and protuberant. The lower part of the bone had apparently suffered as much as the shaft in the process of dwarfed development. The lines running from shaft to condyles were proportionately shortened.

Probably this latter fact accounted for the lack of development of the supinators and flexors of the right arm. The line to internal condyle was a little shorter than that to external condyle. See Plate I, Figs. 1 and 2 for photographs of these cases. Skiagraph of Case A. A. only was obtainable (Plate IV, Fig. 2).

CASE L. S.

Measurements: Weight, 101 lbs.; Height, 5 feet 3¼ inches.

	RIGHT.		LEFT.	
Dynamometer	42	kilog.	38	kilog.
Length of humerus	9	inches.	12½	inches.
Length of ulna	9	"	9	"
Length of radius	8¼	"	8	"
Length of hand	7	"	7	"
Length from anterior superior spinous process of ilium to internal malleolus	31	"	30¼	"
Length of foot	8¾	"	8¾	"
Circumference of arm	8¾	"	8¼	"
Circumference of forearm	7¾	"	7¾	"
Circumference of hand at knuckles (in extension)	7	"	6⅞	"
Circumference at gluteal fold looping to anterior superior spinous process	23½	"	22¾	"
Circumference above knee	11¾	"	11¼	"
Circumference at calf	11¾	"	11	"
Circumference of foot at internal cuneiform and metatarsal articulation	7¾	"	7⅝	"

CASE A. A.—No. II.

Measurements: Weight, 95¼ lbs.; Height, 5 feet 4¼ inches.

	RIGHT.		LEFT.	
Dynamometer	45	kilog.	43	kilog.
Length of humerus (acromion to external condyle)	7½	"	12	"
Length of forearm (external condyle to styloid process)	10	"	10	"
Length of forearm (internal condyle to styloid process)	9½	"	9½	"
Length of hand	7¾	"	7¾	"
Circumference of biceps (largest part)	9¾	"	9¾	"
Circumference of forearm, below elbow	8¾	"	9½	"
Circumference at wrist	5¾	"	6	"
Circumference of hand at knuckles (in extension)	7½	"	7½	"

	RIGHT.	LEFT.
Length, anterior superior spine to internal malleolus	kilog.	32½ kilog.
(Amputation above knee of right).		
Length of foot	"	9½ "
Circumference at gluteal fold, looping from anterior superior spinous process of ilium	"	22½ "
Circumference above knee	"	12½ "
Circumference at calf	"	11⅜ "
Circumference at ankle	"	7½ "
Circumference at internal cuneiform and metatarsal articulation	"	8 "

Chapter IV.

THE WRITER'S CASES OF HYPERTROPHIC FORM OF INFANTILE CEREBRAL PALSY ASSOCIATED WITH EPILEPSY.

Case 1.—E. P. (Plate I, Fig. 3. Plate 2, Fig. 2); female, German; age, 23; no occupation. Admitted to Craig Colony July 15, 1898, suffering from epilepsy. Her convulsions dated from six years of age. Her father died of cancer of the stomach. No alcoholic or tubercular history was attainable. Her mother had rheumatism; a maternal aunt epileptic. She has one brother and one sister who are both healthy. Her mother states that instruments were used during patient's birth, but she sustained no injury at that time and that patient was a healthy, strong child. But she was fretful and crying during teething (at six months of age). Patient began to walk at two years of age and had no serious illness until the age of six.

History of Paralysis.—At six years of age she was suddenly seized with severe and prolonged convulsions, lasting from 24 to 36 hours. Immediately after and dating from this illness, she was paralyzed on right side and unable to use the upper or lower extremity for some time. From this illness to the present time she has had one, two or even three grand mal seizures daily, predominating in paralyzed side. No cause was alleged for the paralytic stroke.

Present Condition.—Patient presents more than the ordinary mental impairment seen in infantile hemiplegics.

She has no education, but is able to do some housework of a simple nature not requiring the use of both hands. It was impossible to perfect skiagraphs for reproduction in this case on account of the persistent mental and physical agitation produced by the X-ray.*

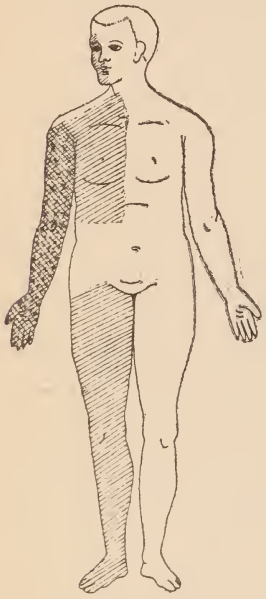


FIG. 6.
Writer's First Case.

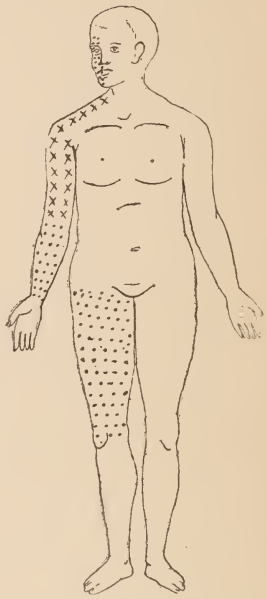


FIG. 7.
Writer's Second Case.

//=parts showing athetosis.
##=parts showing most athetosis.
⠿⠿=parts showing hypertrophy.
××=parts showing most hypertrophy.

*It is interesting to note the behavior of this epileptic patient (E. P.) while effort was being made to obtain a skiagraph of the hypertrophied arm. Contrary to the usual experience an undue emotional state induced a mild state of epilepsy. The patient had been under the rays a few seconds when loss of consciousness without muscular convulsion supervened. The patient seemed to pass into a deep sleep and remained very still for a short time, then a fibrillary tremor occurred on the right side which gradually increased to convulsive jerks of the same side; these latter movements, although not violent, broke the plate. The head was turned to the left and the eyes remained closed throughout the convulsion. For a few seconds after the attack, which lasted one minute, the patient was automatic.

Physical Examination.—Patient in state of good nutrition and suffering from no marked imperfections except the paralytic disorder and its sequent phenomena.

The right side of the body (paralytic side) is in good nutrition and hypertrophic (Text-Figure 6). Some slight vasomotor disturbance is present. The entire right side is in choreiform or athetoid movement not markedly different from ordinary infantile hemiplegia attended by atrophy. It will be seen that the hypertrophic condition is most marked in the upper extremity and that the condition is also apparent in the bony structure as in Kaiser's case.

Again, by the measurements we find the forearm lengthened, while the arm holds the same comparative measurements. Of the two bones of the forearm the radius might be considered the more distal and here we notice the greatest increase in length. We see the comparative increase in length of bone is in the forearm, while the muscular hypertrophy is most marked in the arm. In the hands we find muscles and bones both atrophied on the right side, although the greatest part of the athetoid spasm is in the wrist and hand. The gait is spastic-ataxic and suggests the idea of shortening of the right leg, but measurements do not sustain this suggestion.

Properly speaking, the whole right side is hypertrophic, (paralyzed parts are almost always atrophic) but not really so except in upper extremity, when compared with the left. See Plate I, Fig. 3. The deep reflexes are all exaggerated on the right; cutaneous reflexes (scapular, pectoral and epigastric) are much delayed and diminished in intensity. Right plantar reflex is exaggerated as compared with left. Examination of deep reflexes always creates tremor. Pin prick shows general right side diminution and delay in conduction of sensation of pain. Tactile, heat, and cold sense is slightly impaired. Spasm of muscle is quite marked and efforts to open and shut the hand quickly is done slowly and imperfectly. The hesitation in performing a purposive act does not amount to myotonia. There are no associated movements and patient is left-handed and only uses the right hand occasionally in dressing herself.

The athetoid movement is occasionally typical of true athetosis, especially in foot, but it generally resembles the arythmic coarse and fine tremors of the post-hemiplegic state. Movements can be controlled in a measure, but several attempts are necessary to do so. Less tremor is obtained when attention is called from the parts, and the tremor entirely ceases during sleep. Dynamograph: right 33, left 60.

The loss of power of the right side appears to be dependent upon the flabby and softened state of the muscles of that side and the inco-ordination of their non-volitional activity. (See Text-Figure 6).

E. P.—3.

Comparative Measurements of Right and Left Sides.

Weight, 138 lbs.; Height, 5 feet 3¾ inches.

	RIGHT.		LEFT.	
Dynamographic test	33		60	
Length from acromion process to external condyle	12	inches.	12½	inches.
Length internal condyle of humerus to the styloid process of ulna	9½	"	9½	"
External condyle to styloid process of radius	10	"	9½	"
Circumference of arm; arm flexed on fore-arm at right angles	10¼	"	9½	"
Largest part of forearm in its anatomical position	9¼	"	8¾	"
Circumference of hand at metacarpo-phalangeal articulation	7 7/16	"	7½	"
Length of index finger	3⅝	"	3½	"
Length of second finger	4½	"	4	"
Length of thumb	2⅝	"	2¼	"
Circumference of middle finger at middle part of first phalanx	2¼	"	2⅝	"
Circumference of thigh at gluteal fold	20½	"	20¼	"
Circumference popliteal space	14¼	"	14	"
Circumference of leg at calf	13⅜	"	13½	"
Dorso-plantar circumference	8½	"	8½	"
Circumference of wrist	6	"	5⅞	"
Circumference of ankle	7⅞	"	7⅞	"
Length from ant. sup. spinous process to int. malleolus of ankle	32¾	"	33	"

CASE II.—*Family and Personal History.*—F. W., age 25, female, nativity U. S., single; father died of general paresis at 54, was probably syphilitic, although this was expressly denied by his son. Mother was very nervous, and subject to anomalous attacks of migraine, and had "spells" during pregnancy; patient is second child in family of three—two boys and one girl. The birth of patient was difficult, and labor was instrumental, it being a breech presentation. She was born nevertheless a healthy child.

Between two and three years of age, at dentition, she had a series of convulsions which left the right side hemiplegic. She had two or three slight general spasms when five or six months old, but no ill effects following them. She had croup in infancy, and so did her two brothers.

She had many convulsions after the right palsy at three, which were confined to the paralyzed side. After a few years they became grand mal in type, but began or ended on the right side. No definite aura preceded her attacks.

Automatism frequently followed the epileptic convulsions from the onset of the disease, and occasionally she had a mild attack of mania, in which she showed some violence for a few hours only.

Attacks gradually became more frequent, until as many as eight or ten occurred in four or five days, both by night and day. Patient has never menstruated.

Present Condition.—On admission to Craig Colony patient was found to be a right-side hemiplegic, and measurements proved the whole right side hypertrophic. See Plate II, Fig. 1, also skiagraphs in Plate IV, Fig. 1. *There was no tremor or disorder of motion in paralyzed side.* Patient uses both right and left hand, but commonly the right. (See Text-Figure 7).

F. W.—2.

Comparative Measurements of Right and Left Sides.

Weight, 108 lbs.; height, 4 feet 11¾ inches.

	RIGHT.	LEFT.
Dynamometer	46	50
Dynamometer (average) of twelve tests	42½ inches.	48⅔ inches.
Length from acromion process to tip of middle finger	26½ "	26½ "
Circumference of biceps (forearm at right angles to arm)	10½ "	9¾ "
Circumference below bent elbow (forearm at L)	10 "	9¼ "
Circumference at wrist	6½ "	5¾ "
Circumference behind knuckles	7½ "	7¼ "
Length of middle finger	4 "	4 "
Length of humerus (acromion to ext. condyle; arm bent at L)	11 "	11½ "
Olecranon to styloid process (ulnar side)	9¼ "	9¼ "
Circumference at gluteal fold	19¼ "	19 "
Anterior superior spine to internal malleolus	31 "	30½ "

In Recumbent Position.

Circumference above knee	13½ inches.	13½ inches.
Circumference of calf	13½ "	13 "
Circumference above ankle	7½ "	7½ "
Circumference of arch of foot	8 "	7⅞ "

All deep reflexes were found exaggerated on the right side. Patient shows the feeble-mindedness common to hemiplegics and epileptics.

The peculiar appearance of patient's hands attracted attention at once, and after a most painstaking examination it was found that a mild degree of morphœa or circumscribed scleroderma was present, and was progressive. (See Plate III, Fig. 1). In addition to this most unusual phenomena, patient suffered about four years ago without apparent cause from mortui digiti, (a peculiar type of Raynaud's disease), see Plate III, Fig. 2, of the two last fingers of the right hand. A large trophic ulcer

(gangrenous) of right wrist and several smaller ulcers have formed on the back of the left hand, typical of this disease. Both hands from the elbows down are bronzed similarly to Addison's disease, and present marked cutaneous induration and thickening. Bronzed sclerosed plaques cover the knuckles of the right hand and second finger joints of the same; parallel ridges of scleroses and induration join the plaques on knuckles of first and second fingers of right hand; this is also present to some extent on fingers of left hand. Bronzed indurated patches are found at knees and ankles. Both hands show many other various trophic states aside from those mentioned, giving to the casual observer the idea of leprosy. Mild analgesia and anæsthesia exist in both extremities, most marked in right hand.

All the usual phenomena of a mild type of scleroderma and Raynaud's disease were found in this case.

In going over all the literature upon Raynaud's disease and scleroderma in their varied manifestations I was able to find but one case of scleroderma associated with epilepsy. The literature of scleroderma in general is well known. Lewin in 1895 collected 508 cases, and there are probably between 500 and 600 cases now on record. Spillman, of Paris, reported the only case in all literature of epilepsy with scleroderma. It will be given in abstract in a future paper upon epilepsy, scleroderma and Raynaud's disease.

Raynaud's disease is a more vague condition handled by the neurologist, surgeon and dermatologist, and not being so clear cut a disease, it is doubly difficult to define in reference work. Nevertheless it may be said that it has been seen and fully described in many neuropathic states, especially in insane cases showing trophic disorders and has been noticed in connection with epilepsy in six cases to be given at another time.

The details of two epileptic attacks, out of many of F. W.'s epileptic phenomena witnessed by me, are as follows:

April 3, 1899.—Patient had two severe attacks, which were similar in character; the head was drawn backward, eyes closed, right arm and both lower limbs were straight, with muscles in clonic convulsion; left arm and hand were cold and deeply cyanosed, but not convulsed. When left arm was lifted, it was found to be limp and lifeless, and dropped heavily when released. Clonic convulsion lasted three minutes, the termination was in the right arm; it was then thrown suddenly and violently outward, and as quickly brought back to the patient's side. Stertor lasted several minutes, gradually passing into normal respiration.

April 16, 1899.—Patient had a severe attack while walking across the floor, looked startled, turned her head to the right, and fell to the floor. Convulsion became apparent in the right arm first; then right leg, left arm, left leg, then became general, predominating after a few seconds in the left arm, and finally ending in the right arm, which remained convulsed for several seconds after all other muscular spasm had ceased. Convulsion consisted of both tonic and clonic spasm; stertor lasted about ten minutes, merging into sleep which lasted about twenty minutes.

The points of interest in this last case are as follows:

First.—The hypertrophic state of infantile cerebral palsy in which the face participates to a marked degree.

Second.—The absence of any disorder of motility, such as tremor or athetosis, which explodes the "athetosis theory" as a cause for the enlargement.

Third.—The presence of the circumscribed type of scleroderma and Raynaud's disease, together with palsy and epilepsy, a previously unreported combination.

CONCLUSIONS.

We can urge no new theory to explain the hypertrophic state in these rare cases of infantile cerebral palsies. While evidence is presented here against the prevailing theory of athetosis causing the condition, yet it is to be regretted that definite and sufficient data are not forthcoming to establish a more tenable theory in its place at the present time.

In some instances it is doubtful if a true hypertrophy exists. In others, when pseudo-hypertrophy would seem to be excluded, the power of the enlarged muscles appears to be quite defective. In F. W., of my cases, where no athetosis was ever present, the hypertrophy was greater than in case E. P., and the increased muscular power was proportionate.

While we accept the statement of some that the presence of the hypertrophic state in infantile cerebral palsy is at present an unexplainable pathological freak, yet data must soon be forthcoming which must aid us in solving the problem. As about 70 per cent of all cerebral palsies of children develop epilepsy in later life, from three to twenty years after the original lesion is established, and as the colonization of epileptics is progressing rapidly, thus bringing the adult infantile palsy cases under closer scientific supervision and observation, we see much cause for hope that the etiological pathology of hypertrophic states of cerebral palsies may be elucidated in the near future. The clinical method of physio-pathological study of disorders of mobility must also contribute its share toward explaining atrophic and hypertrophic states in infantile cerebral palsies, and enable us to find just what relationship exists between athetosis and hypertrophy in palsy

cases. If athetosis stands in no causal relation but only as a coincidence, then the hypertrophy is dependent upon some other cause, which I am persuaded is the case.

ACKNOWLEDGMENTS.

It affords me great pleasure to acknowledge in serial order my indebtedness for aid in presenting these contributions.

To the Medical Superintendent of Craig Colony, Dr. William P. Spratling, my thanks are especially due for his encouragement and invaluable suggestions in carrying out these investigations.

The nurses of the colony have also rendered me quite important assistance. By means of their aid I was enabled to have many of my cases watched almost continuously during any interesting set of phenomena, and have the advantage of being able to fill in many gaps which would have otherwise necessarily escaped my own observation.

To the untiring perseverance of my colleague, Dr. E. A. Sharp, is largely due the photographic illustrations and the material furnishing the outline attitude charts in the text of the paper on Exhaustion-Paralysis. Any one who has attempted to catch the most important phases in the fleeting panorama of isolated epileptic paroxysms with the aid of the ordinary photographic apparatus, will fully appreciate the difficulties one has to contend with. A cinematograph, which is about to be used at the Colony, is required for this work and its application in recording the complete phases of the fit ought to disclose many interesting manifestations.

I am much indebted to Dr. Louis A. Weigel of Rochester, N. Y., one of the consulting orthopædic surgeons of the Colony for careful work in taking the skiagraphs presented in the third paper.

Finally, the present affiliation of the scientific work of the Colony with the Pathological Institute of the New York State Hospitals has been of material advantage in counsel and suggestions in the preparation of the study.

References to the Cases.

Gowers: On Athetosis, etc. Medico-Chirurgical Transactions, 1876. (One case).

Bernhardt: Ueber die von Hammond Athetose gennanten symptomen complex. Virchow's Archiv., Bd. 67, (1876).

Bernhardt: Ein neuer Beiträg zur Lehre von der Athetose. Deutsch med. Wochenschrift, Dec. 2, 1876. (Two cases).

Oulmont: De l'athétose. Thèse de Paris, 1878. (One case).

Gaudard: Contribution á l'étude de l' hémiplégie cérébrale infantile, Geneva, 1884. (One case). (Not accessible).

Scheiber: Ueber ein Fall von Athetosis spastica. Arch für Psychiatrie, XXII (1891). (One case). (Not accessible).

Kaiser: Myotonische Störungen bei Athetose. Neurol. Centralblatt, 1897, p. 674. (One case).

Important References without Cases.

Shaw, Clay: On Athetosis or Imbecility with Ataxia. St. Barthol. Hosp. Reports, 1873.

Dreschfeld: De l'athetosé. Rev. mens. de méd. et de chir., 1878, p. 66.

Berger: Athétose. Eulenberg's Real-Encyclopadie, 1885.

Freud und Rie: Klinische Studie über die halbseitige Cerebrallähmung der Kinder. Wien, 1891.

Brissaud: Encéphalopathies infantiles. Charcot Traité de méd. Paris, 1894.

Sachs: Nervous Diseases of Children, 1895, p. 535.

Freud: Die infantile Cerebrallähmungen Nothnagel's Handbuch der specielle Pathogie und Therapie, 1897.

ERRATUM.

For Chapter XI, p. 463, read Chapter VIII.

EXPLANATION OF PLATES.

Plate I.

Figure 1.—Shows case L. S., phocomelus of the right humerus, a shortening of 3½ inches. Left side slightly atrophic from old infantile palsy.

Figure 2.—Shows case A. A. with phocomelus of the right humerus; 4½ inches shorter than the left humerus. No other abnormality in the case.

Figure 3.—Shows the patient E. P. in sitting position, grasping a rigid bar in both hands equally, to enable photograph to be taken. Right arm hypertrophy is well shown. The face is quite typical of such cases suffering from chronic convulsive disorders. The muscular over-action of right side of face is seen here, even under the most perfect mental and physical repose obtainable. Under anæsthesia, measurements showing hypertrophy, remained unchanged.

Plate II.

Figure 1.—Shows full length photograph of case F. W. Right side hemiplegic, with hypertrophy of that side. Hypertrophy most marked in right arm. Right leg one-half inch longer than left. Pronounced sclerodermatous plaques on knees and ankles anteriorly are well shown. No athetosis in this case.

Figure 2.—Full length photograph of case E. P., showing the right side paralytic. The standing of patient is classic of old infantile palsy. Athetosis or post-hemiplegic mobile spasm is very marked in right arm, part most hypertrophic.

Plate III.

Figure 1.—Shows posterior view of hands in case F. W. The sclerodermatous plaques are well shown over the knuckles of the right hand, and also to a slight degree on lines running from knuckles to proximal joint of first two fingers in right hand. Attention is called to the trophic state of the fingers quite typical of the mild form of Raynaud's Disease.

Figure 2.—Shows view of palmar surface of hands in the case F. W. Old scars of Raynaud's affection, resembling anæsthetic leprosy are visible on right hand, especially on joints of the third and fourth fingers.

PLATE IV.

Figure 1.—Show comparative skiagraphs of posterior view of right and left humeri of the writer's hypertrophic palsy case F. W. The hypertrophy of right humerus is very apparent, a fact not demonstrated before by skiagraphs in such cases.

Figure 2.—Show comparative skiagraphs of the writer's case of phocomelus (A. A.); posterior view of right and left humeri shown. Phocomelus of right arm (dwarfed humerus) is very marked.

HYPERTROPHIC PALSY

Fig. 3.

Fig. 2.

Fig. 1.

Fig. 1.

Fig. 2.

HYPERTROPHIC PALSY.

Fig. 2.

Fig. 1

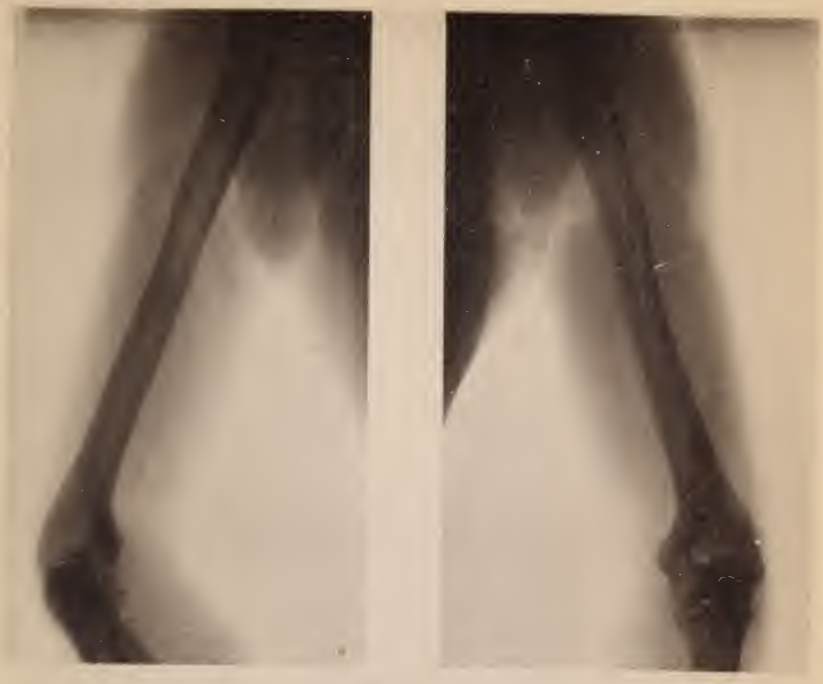


Fig. 1.

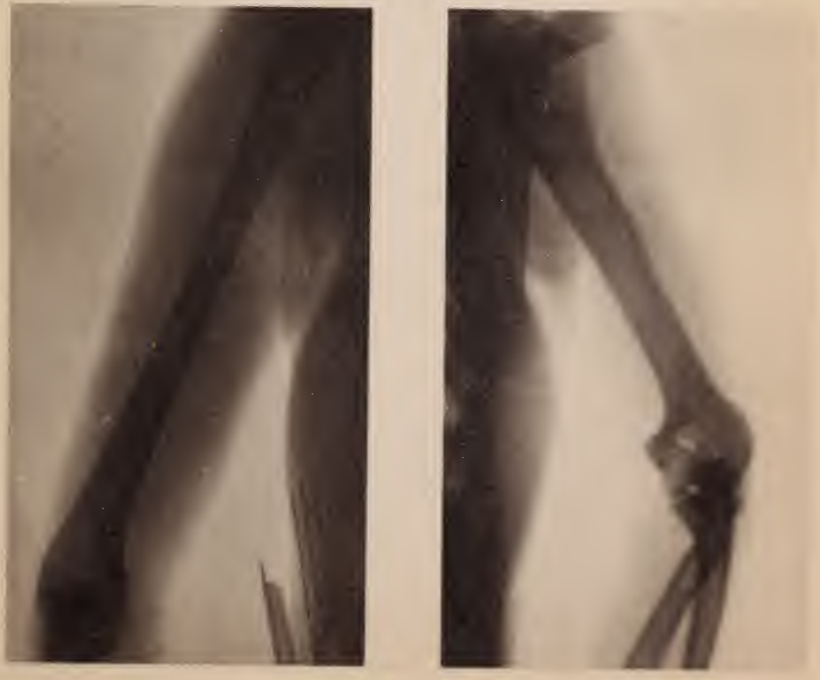


Fig. 2.

ON THE ABSORPTION OF PROTEIDS.

BY P. A. LEVENE AND I. LEVIN.

[*From the Pathological Institute of the New York State Hospitals and the Physiological Laboratory of Columbia University at the College of Physicians and Surgeons, N. Y.*]

I.

In the process of nutrition of the higher animals the first stage is the digestion of the food in the gastrointestinal tract, which is followed by the absorption of the products of digestion.

A derangement of either of these two functions must necessarily lead to depression of the nutrition of all the organs and tissues of the animal body. Therefore the study of the absorption from the gastrointestinal tract has not only a theoretical but also a practical interest.

As regards proteid material it was until a very recent date generally accepted that it was taken up from the digestive tract by the blood system and thus forwarded to the other tissues of the body. Leo Asher was the first to raise objections to the dominating view. He very justly thought that it was based on unsatisfactory experimental evidence. His own investigations led him to the theory that the path of absorption of proteid was the lymphatic system. L. B. Mendel repeated the experiment of Asher and the results he obtained were such as to corroborate the older theory.

The main difficulty in solving the problem seemed to us to lie in the fact that the ingested proteids once past the barrier of the gastrointestinal wall, could not be distinguished from the tissue proteids. To ingest a proteid of

a nature normally not met with in the tissues of the body would permit us to overcome the difficulty. Thus it occurred to us to investigate whether iodoproteids ingested in different parts of the intestinal tract could be detected in the lymph of the thoracic duct.

For different experiments the large intestines and colon, the small intestines or the entire digestive tract were selected as the place of ingestion. The lapse of time between the introduction of the iodoproteid and the beginning of collecting the lymph varied quite extensively in individual experiments. The details will be best seen from the reports.

II.

IODOPROTEID.

Of the iodoproteid employed in the course of this work one part was prepared by ourselves, and the other furnished by Dietrich (Helfenberg), a substance named *a*-eigon.

Our own proteid was prepared in the following way:

Filtered egg-white was treated with an equal volume of a saturated solution of sulphate of ammonia, left over night and filtered. The filtrate, now free of globulins was treated with a solution of HIO_3 and a solution of KI was gradually added until a dark brown ppt. was formed. The entire mixture was being well shaken while the KI was added. The mixture was then left over night in an incubator at 40° C, then filtered and the ppt. suspended in H_2O and dialyzed. It took many weeks before the proteid was free of traces of inorganic I. Towards the end of the purification of the proteid it was dialyzed into 50 per cent alcohol. This proteid did not give Millon's test, and was soluble in alkalies, and contained *no trace* of inorganic I. However, we did not have sufficient time to

prepare all the material required for our experiments, and it was used only for the experiments on the absorption from the large intestines and colon.

For the other experiments the product known as *a*-eigon was used. The analysis of it showed that it contained a considerable quantity of I salt, and it took nearly as much time to purify it as to prepare our own iodoproteid free of I salts. The proteid freed of the latter salts proved to contain I, but was not saturated, as it gave a positive Millon's test, and was insoluble in dilute alkalies. This commercial product was used for the experiment.

III.

METHOD OF THE LYMPH ANALYSIS.

The lymph was collected directly into alcohol, extracted with alcohol until the extracts contained no I. The residue of the proteids was then dried, pulverized, fused in a mixture of one part of $Na_2 CO_3$ and two parts of KNO_3; the fusion dissolved in H_2O, some sulphurous acid was then added, and I tested for by means of chloroform.

IV.

EXPERIMENTS.

Experiment 1, December 13, 1898. Dog; weight about 14 kilos. Fasting 24 hours previous to the operation.

2.30 P. M. The colon was ligated at a distance of about 50 cm. from the periphereal end; a canula inserted, and the colon and rectum washed with normal salt solution. The rectal end was then ligated and 140 cc. of a 10 per cent iodoproteid solution injected. The lymph was collected between 4 and 5.30 P. M. At the end of the operation 60 cc. of solution was found in the loop, but no iodoproteid was found in the lymph.

Experiment 2, December 20, 1898. Dog; weight about 16 kilos, fasting as in experiment No. 1. Operation on the colon and rectum as in No. 1. At 11 A. M. 120 cc. of the 10 per cent proteid solution injected, and at 3 P. M. an additional 80 cc. of the same solution was injected. The lymph which was collected between 4.30 P. M. and 5.30 P. M. did not contain any iodoproteid. The lumen of the intestine, emptied at the end of the operation, contained about 60 cc. of the solution.

Experiment 3, December 23, 1898. Dog; weight 18 kilos; 3 P. M. starvation and operation on the colon and rectum as in experiments 1 and 2; 160 cc. of the solution injected. Lymph collected from 4 P. M. to 5.15 P. M. No iodoproteid in the lymph. At the end of the operation 80 cc. of liquid found in the lumen of the intestine.

Experiment 4, January, 1899. Dog; weight 14 kilos; 2.30 P. M. operation on the colon and rectum; 140 cc. of the iodoproteid injected. Lymph collected from 3.30 P. M. to 5.15 P. M. No iodoproteid in the lymph. About 80 cc. of liquid found in the loop at the end of the operation.

Experiment 5, March 24, 1899. Dog; weight 18 kilos. Thiry's operation performed. March 27, 10 A. M., 60 gr. of the iodoproteid suspended in water injected into the loop. Operation on the thoracic duct performed 3 P. M. Lymph collected until 4.45 P. M. No iodoproteid in the lymph. A considerable amount of the proteid found in the loop.

Experiment 6, April 11, 1899. Dog; weight 15 kilos. Thiry's operation performed. April 13, 11 A. M., 90 gr. of iodoproteid injected into the loop; 3.30 P. M. operation on the thoracic duct performed. Lymph collected until 4.55 P. M. No iodoproteid found in the lymph. Some iodoproteid found in the loop.

Experiment 7, April 18, 1899. Dog; weight 20 kilos. Thiry's operation performed. April 20, 10 A. M., 60 gr. of iodoproteid injected into the loop; 1 P. M. the thoracic duct operated upon. Lymph collected until 5 P. M. No iodoproteid in the lymph.

Experiment 8, April 25, 1899. Dog, weighing about 9 kilos. A gastric fistula had been made by Prof. Curtis several weeks previous; 10 A. M. 60 gr. of the iodoproteid suspended in H_2O was injected into the stomach. At 2 P. M. the thoracic duct was opened and the lymph collected until 5.30 P. M. No iodoproteid was present in the lymph.

V.

CONCLUSIONS.

Thus it is seen that in none of the eight experiments iodoproteid could be detected in the lymph, a fact which may be ascribed to the following different causes:

First, it may be thought that iodoproteid is not absorbed from the gastrointestinal tract at all, but the contrary is evident from the fact that the iodoproteid of the thyroid gland is absorbed, as appears from the beneficial effect of medicinal doses of the gland in myxœdema. Besides this it has been demonstrated by Blum that the artificial iodoproteid is absorbed because he found I in the urine after feeding animals with the iodoproteid.

Second, it may be assumed that iodoproteid simply loses its iodine on digestion, which is also contradicted by the facts recently published by Oswald. Oswald experimented with the artificial digestion of iodoproteid (pepsin and HCl) and found that the I was not released.

Finally, it may be thought that the absorption is so very slow as to make the amount of iodoproteid in the lymph too insignificant to be detected.

But in some of the eight cases the lymph was collected for nearly three hours. We think, however, that this point should be more carefully investigated, and we hope to do so after we prepare a sufficient amount of iodoproteid free of I salts.

As it is at present, our experiments tend to corroborate the older views on the absorption of proteids; they are in accord with the opinion of L. A. Mendel and contradict that of L. Asher.

We wish to express our gratitude to Professor J. G. Curtis and Dr. F. S. Lee of the Department of Physiology of Columbia University School of Medicine, where the operations were performed.

MAY 22, 1899.

REFERENCES.

ASHER AND BARBERA: Centr. f. Physiology, XI.

L. B. MENDEL: American Journal of Physiology, Vol. II, No. II.

OSWALD: Zeitsch f. Physiol. Chemis., XXVII.

EMBRYOCHEMICAL STUDIES.

I.

Some Chemical Changes in the Developing Egg.

By P. A. LEVENE.

[*From the Pathological Institute of the New York State Hospitals and the Department of Physiological Chemistry of Columbia University.*]

I.

INTRODUCTION.

In his remarkable book on general physiology, Max Verworn says: "Der Lebensvorgang beruht in dem Stoffwechsel der Eiweisskörper." I am not certain whether at the present state of science we are justified in making such positive statements that life is only a chemical process. However, it is evident to every biologist that the workings of all mechanisms in which life manifests itself to us, lead to constant wear of those mechanisms or organisms. Biologists have also observed long ago that the living organism possesses a peculiar ability of repairing its constant losses. In fact, there are but very few conditions in the organism when a substance cannot be classified among "the dead," and when the two processes, waste and repair, are not to be noticed. In most conditions of life we can well distinguish these two main functions, dying and growing. And the state of any living organism, its working capacity, its "quality," so to say, depends fully on the relation between these two functions, which Max Verworn calls "biotonus." He further very ingeniously presents the last in form of a fraction A/D, (A = processes of assimilation; D = processes of dissimilation.) Thus the different states of the biotonus might be represented as

$$\frac{A}{D} = 1; \quad \frac{A}{D} > 1; \quad \frac{A}{D} < 1.$$

The significance of this is self-evident. In one case the assimilation and dissimilation are in a state of equilibrium; in the other assimilation predominates; in the third, dissimilation takes the first place. It is further self-evident to any student of biology that none of these processes is a single chemical reaction, that processes of formation, growth, as well as those of decomposition are very complicated; that before the body substance is transformed into final decomposition products, it undergoes many intermediate changes, and before food is assimilated and converted into a part of the body protoplasm, it undergoes numerous transformations. Thus, Verworn presents a general formula of the "biotonus," as

$$\frac{a + a_1 + a_2 \text{ - - - } a_M.}{d + d_1 + d_2 \text{ - - - } d_M.}$$

This mathematical representation of the biotonus is true not only speculatively, but is also in accord with experimental evidence. It should be remarked that physiological chemistry (organic as well as inorganic) began its work, broadly speaking, with analytical experiments; it began by studying the path of transformation of that most complex substance protoplasm, into its final decomposition products, urea, CO_2, ammonia, etc. It first closely followed this path in the living organism, and finally succeeded in imitating the organism, and at the present day we may obtain nearly all the decomposition products met with in the organism, by mere chemical means. But if our knowledge of the process of dissimilation has become quite extensive, we must on the other hand own that the process of synthesis of living substance, even of proteids alone, is as dark to us to-day as it ever has been. And yet nature offers to us conditions when

the growth of the organism is so much predominating over its wear that it seems there ought to be little difficulty in following the organism in its process of growing.

All the highest organisms develop from one single cell, and in many organisms their growth takes place outside of the body of the parent organism. In the animal kingdom, among others, the amphibia and birds belong to the last, and they offer good material for the study of the chemical changes in the growing tissue or organism.

It is singular that in the development of biology, the discoveries of botany nearly always preceded those of the animal biologist, and this has repeated itself again in the study of the relation of chemical changes in the growing or rather developing organism. The work of E. Schulze and his school is remarkable in its results, (and we refer the reader who is interested in the subject, to the original articles), but very little has been done in this direction by the animal physiologist.

The work we are publishing here is the beginning of a series of articles on the chemistry of the developing egg. We think that this general study ought to precede the special study of the development and growth of individual tissues, as muscular, nervous, and glandular tissues, and so on.

Of all the substances most peculiar to the living organisms are the different nitrogenous compounds that take part in formation of the proteid compounds and reappear on the decomposition of the latter.

These compounds may be classified in a general way into two groups: First, those consisting only of C, H, O and N, and second, those in which some other elements, mainly S, P, and Fe (each of them separately, or all together) join the former in the formation of their molecule.

The first group may be again divided into substances with a well defined acid nature, as the monoamido acids, like leucin, and into those of a well defined basic nature, which are very numerous and quite different in their composition.

The second group again may be divided into simple proteids, containing only C,N,H,O and S, and combined proteids as nucleo-compounds, mucin, etc. It is the molecule of the latter compounds that may contain besides C,H,O and N also P and Fe.

The aim of this work was to study the distribution of N between the main groups just enumerated in different stages of the development of the egg, or, to be more precise, we attempted to estimate the quantity of N in the form of compounds not basic by nature, like amidoacid—those in the form of bases and finally those in the form of proteids. Further an attempt was made to ascertain whether in the course of development a new formation of the combined proteids (only the nucleo-compounds were dealt with) was taking place or not. The amounts of ash and water were also estimated.

The material used was the egg of the codfish. It was examined in the following four stages: unfertilized; 24 hours after fertilization; 11 days, and about 20 days after fertilization.

All the material was furnished to us by the courtesy of the U. S. Fish Commission, and we wish to express our indebtedness to Doctor Bumpus and Mr. Locke, who were kind enough to supply us with fish eggs. It was only through their kind assistance that this work could be carried out.

II.

METHODS.

Total nitrogen was determined, after the material was dried to constant weight at 105° C, by Kjedahl's method. The nitrogen in the form of monoamido acids and related compounds was estimated by the following method:

The dry substance was extracted for 24 hours with 0.2 per cent HCl solution. The mixture was then treated with phosphotungstic acid, and after standing 24 hours the ppt. containing the insoluble part of the tissue and the phosphotungstic ppt. digested by Kjedahl's method (K_2SO_4 and C_U SO_4 used for digestion).

For estimation of the proteid nitrogen, the substance was first extracted in a Kjedahl's digestive flask, for 24 hours with boiling alcohol, then washed with ether and alcohol, and treated with boiling water and a few drops of acetic acid for about 10 hours and with cold water for about 10 hours again, and then the N estimated by Kjedahl's method. (All the extracts were tested for proteids. The results were negative).

To study the changes in the quantity of nucleo-compounds, and nucleo-bases, the eggs were extracted with cold and hot alcohol, then dried in air, pulverized, again extracted with hot alcohol, cold and hot ether, again dried, first in air, then at 105° C.

To estimate the nuclein-bases, the substance was heated on a water-bath in a flask with a return condensor with 2 per cent H_2SO_4 for about 10 hours. The acid was partly neutralized by Ba (OH_2), the filtrate concentrated, the silver salts of the nuclein-bases obtained, and weighed as such.

Another part of the same material which was used for

determination of the nuclein-bases was digested with pepsin-hydrochloric acid during one week, changing the digestive fluid every second day. The residue was then washed with water, until the latter gave a negative Biuret reaction, and contained no HCl.

It was then washed with alcohol, ether, dried and weighed.

To ascertain whether the residue was really a nuclein or a substance rich in nucleins, the P was estimated; but only in one case, as in the other two the quantity was not sufficient for a satisfactory P estimation.

III.

RESULTS OF THE ANALYSIS.*

We present all the results in short tabular form.

I.—H_2O AND ASH DETERMINATIONS.

	Subst.	Dry Subst.		Ash.	
	In gr.	In g.	In Per Cent.	In grm.	Per Cent.
F-O	9.7612	0.5737	5.33	0.058	10.09
F-I	8.2201	0.4760	5.20	0.648	17.17
F-II	7.06005	0.564	7.98	0.099	17.55
F-III	8.097	0.5315	6.31	0.1045	19.66

II.—DISTRIBUTIONS OF N.

	Subst.	Total N in grm.	Per Cent.	Per Cent.
F-O	0.5405	0.059568	11.01	
				10.90
	0.403	0.0438	10.80	
F-I	0.3914	0.039858	10.16	
				9.96
	0.4299	0.042048	9.77	
F-II	0.2985	0.033288	11.15	
				11.22
	0.3225	0.036354	11.29	
F-III	0.318	0.029346	9.52	9.52

* Fo=unfertilized; Fi=24 hours after fertilization; Fii=11 days after fertilization; Fiii=20 days after fertilization.

III.—N in Phosphotungstic Ppt.=Proteids+Bases.

	Substance.	Grms.	Per Cent.	
F-O	0.3670	0.03066	8.32	
				8.50
	0.2956	0.02628	8.88	
F-I	0.1791	0.014016	7.82	
				7.83
	0.3296	0.025842	7.84	
F-II	0.2855	0.024528	8.52	
				8.67
	0.3366	0.029784	8.85	
F-III	0.2251	0.021462	9.53	9.53

IV.—Proteid N.

F-O	0.1650	0.012264	7.43	
				7.29
	0.2940	0.020824	7.15	
F-I	0.5267	0.028470	5.40	
				5.33
	0.5504	0.028808	5.26	
F-II	0.5535	0.041610	7.52	
				7.27
	0.654	0.04599	7.03	
F-III	0.2575	0.01752	6.84	6.84

V.

	F-O		F-I	
	Per cent to Dry Subst.	Per Cent to Total N.	Per Cent to Dry Subst.	Per Cent to Total N.
N in Monoamido compounds	10.90—8.60 =2.30%	21.10	9.96—7.83= 2.13	21.37
N in form of bases....	8.60—7.29 =1.31%	12.07	7.83—5.33= 2.50	25.10
N in form of proteids.....	7.29	66.00	5.33	53.57

	F-II		F-III	
	Per Cent to Dry Subst.	Per Cent to Total N.	Per Cent to Dry Subst.	Per Cent to Total N.
N in Monoamido compounds	11.22—8.67= 2.55	22.72	9.52—9.53 =—1	0
N in form of bases....	8.67—7.27= 1.40	12.48	9.53—6.84= 2.69	28.25
N in form of proteids.....	7.27	64.79	6.84	71.84

VI.—Digestive Experiments.

	Subst.	Residue in gr.	In Per Cent.
F-I	2.0442	0.0428	2.08
F-II	1.698	0.0570	3.35
F-III	1.7767	0.1297	7.24

P.—Determination in the residue of F-III: 0.137 gr. of the residue.

MgP_2O_3=0.014 gr. P=2.65%.

VII.—Determination of the Nucleo-Bases.

	Subst.	In gr.	In Per Cent.
F-O	1.8611	0.0022	0.12
F-I	2.0227	0.0438	2.16
F-II	1,519	0.0325	2.14
F-III	1.2132	0.0455	3.75

IV.

General Remarks.

I think it would be premature to draw any very broad conclusions for the little work done for the present. Such conclusions should be deferred until the data have increased considerably.

The results of this work, however, tend to indicate that in the developing egg the processes of synthesis are preceded by those of decomposition (consult Table V). In the first stage after fertilization the proteids diminish in quantity; basic nitrogenous substances are formed at their expense. Later the basic substances decrease in quantity and proteids grow. Whether the molecules of those proteids are formed from the basic substances will be investigated in the future.

It is also seen that the character of the proteids is changed during the development of the egg, the combined proteids as we may term them, such as nucleoproteids,

increase greatly in quantity. The importance of mineral salts for the formation of tissues can be illustrated by the increasing quantity of mineral substances in the egg in the course of its growth.

I take occasion to acknowledge my indebtedness to Professor Chittenden for all the kindness shown by him to me while I was engaged in this work in his laboratory.

MAY 22, 1899.

BIBLIOGRAPHY.

A. TICHOMIROFF: Chemische Studien über die Entwicklung der Insecteneier. Zeitsch. f. Physiol. Chemic., IX, 578.

A. KOSSEL: Weitere Beiträge zur Chemie des Zellkerns. Zeitsch. f. Physiol. Chemic., X, 248.

ON THE EVIDENCE OF THE GOLGI METHODS FOR THE THEORY OF NEURON RETRACTION.—ABSTRACT.*

[*From the Pathological Institute of the New York State Hospitals*].

BY RICHARD WEIL AND ROBERT FRANK.

In the following paper a brief statement is given of the results of studies by the authors during the past three years in the Pathological Institute of the New York State Hospitals in connection with the theory of neuron retraction. All the histological evidence which has hitherto been advanced in support of the theory has been based upon the Golgi method; the object of the present research is an experimental investigation of the validity of certain histological changes in the processes of the nerve-cell, supposed to be indicative of neuron retraction.

The literature of the subject is extensive and will not be detailed in this place; a complete résumé is furnished by W. Ford Robertson in the last number of the English magazine "*Brain*." In brief, neuron retractility is by the majority of observers regarded as a function of the protoplasmic processes, and is best manifested by the pyramid cells of the cerebrum and the Purkinje cells of the cerebellum. Two changes are said to occur: First, the appearance of localized swellings, or "varicosities," along the course of the dendrites, and, second, the disappearance of

* The paper of which this is an abstract was finished and handed to the editors June 5, 1899, and is arranged for publication in a series of articles on the subject of Neuron Retraction in these ARCHIVES, Vol. III, Nos. 1 and 2. An abstract of the paper was presented at the New York Pathological Society, November 8, 1899, with a lantern demonstration comparing the plates accompanying the article with drawings from the papers of Soukhanoff, Lugaro, Odier and others.

the gemmules, or dendritic spines, at the site of these swellings. The two phenomena are supposed, except by Lugaro, to be correlated: the spinous processes withdraw into the body of the dendrite, and, by so doing, produce a localized swelling. Human and experimental material of the greatest diversity has been investigated—of the former, brains of diphtheria, typhoid, insolation, etc., of the latter, brains of animals poisoned by arsenic, lead, morphine, strychnine, chloroform, tuberculosis, hydrophobia, experimental strumapriva, "experimental uræmia" and so forth. The method employed has always been the rapid Golgi (or Cajal) method, except in the case of certain recent work of Lugaro's, which has employed Cox's modification of the corrosive method. Criticism of the conclusions above stated has not been lacking. Both gemmules and varicosities are by some authorities considered to be artifacts. By others, *e. g.* Lugaro, while their occasional authenticity is admitted, a variable proportion of their number is attributed to post-mortem processes; Lugaro asserts that the only form of the Golgi method which does not exaggerate the true number of varicosities is the Cox modification.

In this investigation, four forms of the Golgi method were made use of, namely, the rapid, mixed, and slow modifications of the bichromate-silver method, and the Cox modification of the corrosive method. The number of animals used was forty-three. There were five cases of human material, three adults and two fœtuses; one dog, and thirty-seven rabbits. Of the rabbits ten were normal; of the remainder two were poisoned by morphine, one by strychnine, four by chloroform, and the rest by the injection of hypertoxic urine or serum. Nine of the rabbits were treated uniformly according to four methods, the

three bichromate-silver modifications and the Cox; two were treated according to the rapid Golgi method and the Cox; the rest according to the rapid, mixed, or slow procedure alone. In all, 342 pieces were sectioned. The cerebral cortex alone was studied.

The conclusions reached are the following:

1.—The same material, when treated by different methods, yields different results. The nature of the differences in case of each kind of material is as follows:

All material treated according to the slow method of Golgi, shows, as a rule, an almost absolute freedom from varicosities; varicose cells occasionally occur, but with a relative frequency which is perhaps not greater than a fraction of one per cent of the total number of pyramid cells impregnated. Exceptionally, a large proportion of varicosities occurs.

The mixed method and the rapid method may be considered together; these two methods yield practically similar results as regards the varicosities and the gemmules. The gemmules are almost invariably present and generally regular, provided the dendrites have taken the impregnation. The varicosities occur in variable proportions, although their frequency regularly is greater, and almost always very much greater, than is the case in the slow method. In some sections, almost every dendrite is varicose, in others, hardly any.

In the Cox method, a fair amount of varicoseness is generally present at any stage of fixation. Gemmules are almost universally present and regular.

2.—The above results are independent of the nature of the material, whether normal or toxic. Normal material, as well as toxic, is, as a rule, free from varicosities when treated by the slow method. Normal material, as well as

toxic, exhibits a variable amount of varicosity, when treated by any of the other three methods which we have used. We find that it varies within exactly the same limits as the abnormal, that every degree of varicoseness can be illustrated with equal freedom from either, and, finally, that it is impossible for an unprejudiced observer to differentiate or distinguish between the two kinds of material.

3.—The same material does not yield constantly identical results, when treated by one and the same method. Pieces from the same animal, when immersed in the same fluids of the slow, mixed, rapid, or Cox method, may illustrate the extremes of varicoseness produced by that method.

The above conclusions seem to demonstrate that the varicosities are to be regarded as artifacts of the Golgi method.

612.01

THE CHEMICAL RELATIONSHIP OF COLLOID MUCOID AND AMYLOID SUBSTANCES.*

(A Preliminary Communication).

By P. A. LEVENE.

[*From the Pathological Institute of the New York State Hospitals.*]

The morphological study of diseased organs revealed frequently the presence in the cells and tissues of substances which do not occur in the same places under normal conditions. The nature of the substances could be detected by microscopical investigation only very rarely. The microscopical technique was inadequate to disclose the chemical nature of most of the "pathological substances," and they were then identified by their physical properties. And yet only a thorough knowledge of their chemical constitution could elucidate the process of their formation and their relationship to the normal cell constituents.

The substance predominating over every other one in the protoplasm and most peculiar to it is one of a proteid nature. It is therefore natural to expect the most changes in the proteids of the tissues, when the latter are affected by a morbid process. Such is in fact the case. Pathologists have described several forms of pathological transformations of proteids in tissues under the name of coagulation necrosis, amyloid, colloid, mucoid, hyalin substances, etc. Originally but one distinction between the latter substances and the physiological proteids was detected. While the normal proteids were found to be in the tissues in a state of solution the "pathological" ones were coagulated. In all other respects they were

*Abstract of a paper read before the New York Pathological Society, December 13, 1899.

similar to any proteid material. Amyloid, colloid, mucoid, hyalin substances were for certain physical properties classified under a special group of "colloidal" substances.

The studies on mucin and allied substances, however, soon disclosed that the latter were not simple proteids, that the proteid was combined in them with a reducing substance, a "carbohydrate" or "animal gum."

Thus mucoid and colloid material had to be regarded as substances distinct from the other colloidal substances — amyloid and hyalin.

Later it was discovered that amyloid was also not a simple proteid. As mucin and mucoid so amyloid material contained a substance capable after certain treatment of reducing Fehling's solution. There was, however, a pronounced difference between mucoids and amyloid, while in the former the "animal gum" was supposed to be combined directly with the proteids, it was in the latter combined indirectly by the aid of sulphuric acid. In a word, in amyloid, the substance capable of reducing Fehling's solution was described as chondrotin-sulphuric acid.

On the ground of the latter discovery three different forms of colloidal substances had to be established.

1. Mucoid and colloid.
2. Amyloid.
3. Hyalin.

However, certain chemical and tinctorial properties of mucin justified, to my mind, the supposition that this substance must have contained in its molecule an acid radical. To find the acid radical of mucin was the object of this investigation.

The work was begun on tendo-mucin, and it was soon found that similarly to amyloid the mucin was a com-

pound of proteid and of an ethereal sulphuric acid. Further it was found that similarly to chondrotin-sulphuric acid the ethereal-sulphuric acid of the mucin was nitrogenous, and that it yielded similarly to the former, chondrosin.

With the same object in view submaxillary mucin and colloid of a colloidal carcinoma were analyzed. These two substances were also found to be compounds of a nitrogenous ethereal sulphuric acid.

Thus, it seems that the acid radicals of amyloid, colloid and mucoid substances are very similar to each other. The investigation into the question whether the substances are identical or only similar is now in progress.

THE SEQUENCE OF CHANGES IN THE OPTIC CHIASM PRODUCED BY ACROMEGALIA, AS EXEMPLIFIED IN THREE CASES.*

BY WARD A. HOLDEN.

[*From the Pathological Institute of the New York State Hospitals*].

In Vol. I, No. 4 of these ARCHIVES I described a case of excessive distortion of the optic chiasm in acromegalia. Since then I have had opportunity to examine two distorted chiasms in acromegalia, and also the skull of the case previously described.

The specimens from these three cases form a series so instructive that we may generalize from them and obtain a definite conception of the entire course of changes which the chiasm seems regularly to undergo in acromegalia. These specimens are of the more importance because in the autopsy reports hitherto published the chiasm usually has been neglected entirely, or if mentioned there has merely been a note to the effect that the chiasm was or was not flattened.

It is proposed, first, to present a brief description of each of these cases in so far as relates to our present inquiry, and then to consider the sequence of changes which take place in the chiasm, and the way in which they are brought about.

CASE I.†—The patient, a woman of 65, died from uræmia and pulmonary congestion in the Almshouse hospital, in the service of Dr. Pearce Bailey. Enlargement of the extremities and head had been noticed for five years. No history of disturbance of vision was obtained. On autopsy made by Dr. Bailey, the pituitary body was found en-

* Read before the Section in Ophthalmology of the New York Academy of Medicine, January meeting, 1900.

† The case was reported in full by Dr. Bailey in the *Phila. Med. Jour.*, April 30, 1898.

larged, weighing 8 grm. and measuring 2.2 cm. in transverse and 1.6 cm. in antero-posterior diameter. The adjacent bone was not eroded and there were no evidences of pressure on the surrounding nerves. Several months later Dr. Bailey was kind enough to give me the base of the brain, which had been hardened in alcohol. After stripping off the membranes the posterior portion of the chiasm appeared somewhat flattened, with compression of the inner portions of the adjacent optic tracts. (Fig. 1, Plate I). Sections through the entire chiasm disclosed a marked difference in thickness between the anterior and posterior portions of the chiasm. After having remained so long in alcohol the chiasm could not well be stained by Weigert's hematoxylin method, but van Gieson's picro-acid-fuchsin stain revealed no marked atrophy of the fibres and no signs of interstitial neuritis.

Case II.—M. F., a woman of 51, died from lobar pneumonia and chronic nephritis in Bellevue Hospital, July 21, 1899, the day after admission, in the service of Dr. Alexander Lambert. The autopsy was made by Dr. LeWald. To these gentlemen and to Dr. Stone, the house physician, I am indebted for the brain and eyes, and for the history of the patient.

The case was one of advanced acromegalia. The enlargement of the extremities had been noticed for more than ten years before the patient's death. Owing to sensitiveness in regard to her appearance she had avoided hospitals and clinics and we could not determine whether her eyes had ever been examined. The relatives had noticed, however, a squint of the right eye; and although she had never worn glasses, in the last months of her life she was obliged to hold a book very close in order to read.

The drawing Fig. 2, Plate I, was made immediately after the removal of the brain. The very greatly enlarged pituitary body, which was smooth, soft and fluctuating, then measured 3.3 cm. in antero-posterior and 2.7 cm. in transverse diameter and almost completely filled the area included within the arterial circle of Willis. The optic

nerves, small and flattened, emerged on either side between the pituitary body and the anterior cerebral artery, and the only trace of chiasm visible was a thin, almost membranous strand of tissue which stretched over the anterior surface of the pituitary body from one optic nerve to the other, and rested on the anterior cerebral arteries and the anterior communicating artery. All the vessels of the circle of Willis were atheromatous and contained irregularly distributed chalky deposits.

After the pituitary body had been removed a deep depression in the base of the brain was disclosed and the chiasm appeared to be entirely destroyed except for the anterior membranous strand just mentioned. The optic nerves were crushed, distorted, and almost severed by the compression effected mainly by the enlargement of the pituitary body and undoubtedly to some extent by the atheromatous anterior cerebral arteries. The optic tracts, small and imperfectly outlined anteriorly, further back curved round the peduncles in a normal manner.

After lying three days in 10 per cent solution of formol, the optic nerves were put into Müller's fluid, later cut in celloidin, and the sections stained by Weigert's hematoxylin method. The right nerve was found to be completely atrophic, while the left was irregularly atrophied, particularly in its central and upper-inner portions (see Fig. 5, Plate I). The retina of the right eye, stained with hematoxylin-eosin, showed a complete degeneration of the nerve-fibres and ganglion cells. There was no evidence in the nerve head of previous inflammation.

Case III.*—The patient, a woman of 44, died of acute nephritis at the Montefiore Home, Nov. 19th, 1898. Dr. Isaac Adler had reported the case as one of well-marked acromegalia ten years before. The autopsy was performed by Dr. Harlow Brooks. Through the kindness of these gentlemen and Dr. Fraenkel, the medical director, I was able to examine the visual tract and skull.

* The examination of the visual tract was reported in full in Vol. I, No. 4. of these ARCHIVES.

According to the records of the Montefiore Home, twenty months before her death the patient had R. v. = $\frac{20}{40}$, L. v. = $\frac{20}{30}$; R. field contracted on nasal, superior and inferior sides to 35°, and on temporal side to 60°; L. field slightly contracted; optic discs pale. Later the acuteness of vision diminished somewhat and the fields became more contracted, but repeated examinations by Dr. Fraenkel failed to reveal bitemporal hemianopsia. Unfortunately the extent of the color fields was not determined.

The pituitary body measured 3 cm. in its greatest diameter, the antero-posterior, and 2.7 cm. from above downward. The chiasm had been greatly flattened out and its median portion forced upward against the base of the brain until each optic nerve appeared to be continuous with the tract of the same side only (Fig. 3).

Anteriorly a thin process extended inward from the right nerve, and a similar thicker process from the left nerve was covered by a square placque of tissue that proved to be capsule of the pituitary body, which had become adherent here and had been detached when the pituitary body was removed.

The brain was hardened in formol for two weeks and then put into Müller's fluid. The process from the right optic nerve proved to consist of connective and glia tissue from which all nerve fibres had disappeared. The middle and posterior portions of the chiasm were greatly stretched laterally and compressed. Still, a modified Weigert's stain showed that many normal fibres of the crossing bundles still remained. The optic nerves just in front of the chiasm were atrophied, chiefly in their inner-lower portions (Fig. 4). The tracts were moderately atrophic. The nerve-fibre and ganglion-cell layers of the retina had not suffered to an appreciable extent. The atrophy had not extended posteriorly beyond the basal ganglia; and the optic radiations, stained by Weigert's method, and the cortex about the calcarine fissure, stained by Nissl's method, appeared normal. No signs of inflammation were found anywhere in the visual tract.

The base of the skull in this case was marked by great

coarseness and irregularity (Fig. 7). The pituitary fossa was enlarged in all diameters,* its antero-posterior diameter being nearly 2 cm. This, it will be noticed, is less than the antero-posterior diameter of the pituitary body after removal, but the latter was soft and fluctuating and this discrepancy seems due to the distortion of the gland after its removal from the bony cavity. The floor of the fossa had been eroded at three points, exposing the cancellous bone. The optic foramina had been forced far apart, and the anterior wall of the fossa with the olivary process had been greatly flattened and pushed forward.

The essential pathological process of acromegalia must now be considered to be glandular hypertrophy of the prehypophysis cerebri. Any considerable enlargement of this gland must necessarily be followed by yielding of the bony walls of the pituitary fossa in which it lies, or by compression or displacement of the optic chiasm which covers it. Thus it happens that some degree of distortion of the chiasm is found in the majority of cases of advanced acromegalia, and the nature of these distortions, hitherto neglected, is certainly of interest.

The three cases here described seem to me to show definitely the mechanism and the sequence of the changes that the chiasm usually undergoes. A seeming peculiarity in the course of these changes in the chiasm is made clear when we take into consideration the distortion which the bony pituitary fossa undergoes in acromegalia. Normally, the anterior portion of the chiasm lies above the olivary process of the sphenoid bone, just in front of the pituitary fossa, and the optic nerves, running to the optic foramina, also lie above the bone, in the optic groove (see Fig. 6). The infundibulum of the pituitary body passes

* For a careful study of measurements of the pituitary fossa, see Hrdlicka, Dimensions of the Normal Pituitary Fossa, these ARCHIVES, Vol. 1, No. 4.

down behind the chiasm and expands into the body proper which fills the pituitary fossa, lying beneath the posterior portion of the chiasm. While the other relations are normal, an upward pressure of the pituitary body can affect the chiasm in its posterior portion only. But with the pushing forward of the anterior wall of the fossa and the flattening of the olivary process that eventually result from the progressive enlargement of the pituitary body, the anterior portion of the chiasm loses the bony floor upon which it has previously rested and it then becomes subject to the encroachment of the pituitary body.

We find, also, that with the general enlargement of the pituitary fossa, the optic foramina are forced farther apart, and this tends to increase the lateral spreading of the chiasm which the pressure of the enlarged pituitary body causes.

There may be a considerable enlargement of the pituitary body in acromegalia without any distortion of the chiasm. This has been noted frequently in autopsy reports and I have, myself, seen it once. The initial stage of the distortion is doubtless that found in Case I in which the optic tracts were compressed from within and the chiasm from behind and below, the posterior portion of the chiasm being much thinned. (Fig. 1). The pituitary body here was only moderately enlarged, and its lateral diameter was greater than its antero-posterior diameter, the fossa probably not having been much enlarged.

In the other cases the pituitary body was much larger, its greater diameter being the antero-posterior, and it is evident that the posterior and middle portions of the chiasm were first compressed and forced upward, hollowing out a cavity in the base of the brain, while the anterior

portion of the chiasm, spared at first and separated from the rest of the chiasm, later was arched directly forward, when the olivary process of the sphenoid bone had become flattened and pushed forward.

In Case III the main portion of the chiasm had been flattened until it covered an area over 2 cm. square, and each optic nerve, in the gross specimen, appeared to be directly continuous with the optic tract of the same side only. (Fig. 3).

In Case II the chiasm appeared to have been entirely cut through except for a thin anterior bundle, arched forward, which was on the point of rupturing in its middle. (Fig. 2). In Case III this anterior bundle had already ruptured and was represented only by an atrophic process extending inward from each optic nerve.

These, then, are the gross changes which the chiasm may regularly be expected to undergo in acromegalia:

First, the posterior portion of the chiasm is compressed by the pituitary body. Following this, the posterior and middle portions of the chiasm are flattened and forced upward, and thus separated from the anterior portion which is protected by the bone beneath it. Later, with this tilting upward of the chiasm posteriorly and the forcing forward of the anterior wall of the pituitary fossa, the anterior portion of the chiasm is encroached upon by the pituitary body and arched directly forward. Finally the chiasm may be severed completely.

We are interested, also, in learning what fibre bundles of the chiasm are affected and what changes are produced in the visual field. The chiasm in acromegalia is, generally speaking, compressed between the base of the brain and the soft encapsulated pituitary body, which never penetrates the chiasm as it may when it is the seat of

malignant growths. The pressure, therefore, is generally diffuse, and the degeneration which is brought about in the chiasm need not always be at the point of contact with the pituitary body. Furthermore, with this diffuse pressure large portions of the chiasm may have their function interfered with, but oftentimes only slightly, so that the resulting defect in the field is not absolute but for colors or pale grays alone.

In Case I the bundles directly compressed would seem to comprise principally those fibres making up the commissures which run from the basal ganglia of one side through the tracts and chiasm to the other side. Destruction of these commissures need not affect vision.

In Case II, besides the apparent cutting through of the chiasm in the median line, the optic nerves were compressed directly between the pituitary body and the rigid anterior cerebral arteries. Therefore it was not surprising that the right nerve was found to be completely atrophic, and the left nerve atrophic in its central and its inner-upper portions.

In Case III, with moderate concentric contraction of the visual fields, it was found that, in spite of the lateral stretching of the chiasm and the compression of its median portion, a certain number of the crossing fibres were still preserved, showing that the optic-nerve fibres offer great resistance to stretching and compression, provided that the offending body be soft and of slow growth. In this case the atrophy of the optic nerves just anterior to the chiasm was chiefly in their inner-lower quadrants.

It is evident that there may be considerable differences as respects the location of the atrophic bundles of fibres in the chiasm.

If the pituitary body becomes enlarged symmetrically,

it may be expected that the chiasm will be compressed posteriorly and flattened out laterally. With a diffuse pressure on the chiasm in its entire lateral extent there will probably be concentric contraction of the visual fields; but if the median portion of the chiasm is the more compressed, as is frequently the case, the crossing fibres in the chiasm will be those most interfered with, and there will result a more or less typical bi-temporal hemianopsia.

If, however, one side of the pituitary body enlarges more rapidly than the other, for a time one tract alone may be compressed, causing homonymous hemianopsia.

Again, the atrophy in one optic nerve may differ from that in the other because one nerve has been pressed against a rigid anterior cerebral artery instead of against the soft brain substance, as was seen in Case II.

Furthermore, Broca believed that the optic nerves might be compressed by a narrowing of the optic foramina. In Fig. 7 it is seen that the optic foramina are, indeed, diminished in size, but in this case the optic nerves were already slightly atrophied and reduced in diameter, and they were not compressed by the bone.

Finally, in view of the fact that there are frequently signs of inflammation of the optic nerves, amounting at times to choked disc, the nerve fibres may be compressed as a result of interstitial inflammation, or the compression of the papillary arteries may lead to the degeneration of the ganglion cells of the retina and ascending atrophy of the nerve. In none of the three cases here described, however, were any microscopic signs of neuritis discovered.

If we turn to the literature, we find that nearly 200 cases of acromegalia have been reported, and that visual disturbances have been noted in about one-half of them. In over 50 per cent of the cases with disturbance of vision,

there has been concentric contraction of the visual field with diminution of central acuteness of vision; in somewhat less than 50 per cent there has been bi-temporal hemianopsia, absolute or for colors only, with or without some contraction of the nasal halves of the fields; in half a dozen cases there has been homonymous hemianopsia, absolute or for colors only; and in one case there was found binasal hemianopsia.

The type of contraction of the field may, of course, change as the pituitary body grows larger. Thus, if there is at first a homonymous hemianopsia from pressure on one tract, the chiasm itself will soon become involved, and that eye which had previously lost its nasal field will now lose its temporal field also.

Again, when at first there has been bi-temporal hemianopsia, later the nasal halves of the fields may be lost, either by slow concentric contraction or by the more rapid loss of individual quadrants.

There is no special time for the appearance of visual symptoms in acromegalia. These come on, occasionally, soon after the enlargement of the extremities is noticed, but usually not until years after, and the disease may exist for ten or fifteen years without the appearance of any visual disturbance whatever.

EXPLANATION OF THE FIGURES ON PLATE I.

Figure 1.—The chiasm of Case I after removal of the pituitary body.

Figure 2.—The chiasm of Case II with the pituitary body in position.

Figure 3.—The chiasm of Case III after removal of the pituitary body. Bosse, del.

Figure 4.—The right optic nerve of Case III, near the chiasm, seen from before. Weigert's hematoxylin stain. Atrophy most marked in the infero-nasal quadrant of the nerve. From a photograph.

Figure 5.—The left optic nerve of Case II, near the chiasm, seen from before. Weigert's hematoxylin stain. Atrophy most marked in the centre and in the supero-nasal portion of the nerve.

Figure 6.—Pituitary fossa of a normal woman.

Figure 7.—Pituitary fossa of Case III. The fossa is enlarged in all directions, the olivary process is flattened, and the optic foramina are separated widely.

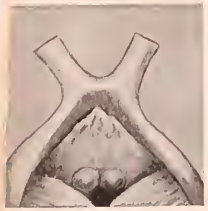


Fig. 1.

Fig. 3.

Fig. 6

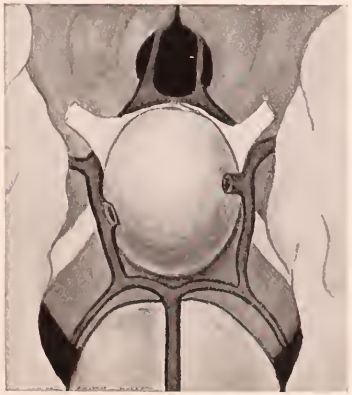


Fig. 2.

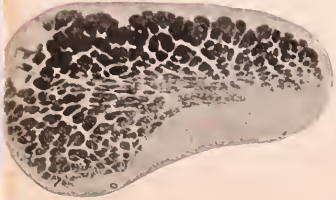


Fig. 4.

Fig. 5.

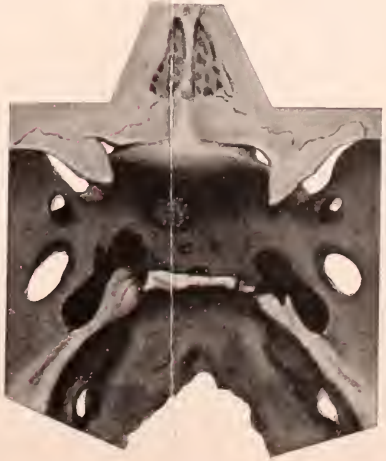


Fig. 7.

HOLDEN DEL.